Earlier versus later cognitive event-related potentials (ERPs) in attention-deficit/hyperactivity disorder (ADHD): A meta-analysis

Kaiser, Anna ; Aggensteiner, Pascal-M ; Baumeister, Sarah ; Holz, Nathalie E ; Banaschewski, Tobias ; Brandeis, Daniel

Abstract: The current meta-analysis summarizes relevant literature on earlier (P100, N100, P200, N200, ERN/Ne) versus later (P300, Pe, CNV) cognitive Event-Related Potential (ERP) differences between children, adolescents, and adults with Attention-Deficit/Hyperactivity Disorder (ADHD) and without ADHD (non-ADHD). Furthermore, the heterogeneity in previous research is addressed by analyzing potentially relevant demographic and methodological moderators (age group, IQ, medication, comorbidity, task, cognitive function, modality, inter-stimulus-interval, number of electrodes). Via database search 52 relevant articles were identified including n = 1576 ADHD and n = 1794 non-ADHD. Using multilevel-models, pooled effect sizes were calculated. For earlier components, individuals with ADHD showed shorter Go-P100-latencies than non-ADHD. For later ERPs, individuals with ADHD showed smaller Cue-P300-amplitudes, longer Go-P300-latencies, smaller NoGo-P300-amplitudes, longer NoGo-P300-latencies, smaller CNV-amplitudes, and smaller Pe-amplitudes. The substantial heterogeneity identified for most of the ERP components could be explained by the demographic and methodological moderators of interest. This meta-analysis identified relevant moderate group differences ($-0.32<d<-0.57$), mainly regarding later cognitive ERPs. Nevertheless, results are characterized by substantial heterogeneity and the moderate effect sizes ($d<0.6$) limit the use for clinical application.

DOI: https://doi.org/10.1016/j.neubiorev.2020.01.019

Neuroscience and Biobehavioral Reviews 112 (2020) 117–134

Contents lists available at ScienceDirect

Neuroscience and Biobehavioral Reviews

journal homepage: www.elsevier.com/locate/neubiorev

Earlier versus later cognitive event-related potentials (ERPs) in attention-deficit/hyperactivity disorder (ADHD): A meta-analysis

Anna Kaiser[a,*], Pascal-M. Aggensteiner[a], Sarah Baumeister[a], Nathalie E. Holz[a], Tobias Banaschewski[a], Daniel Brandeis[a,b,c,d]

[a] Department of Child and Adolescent Psychiatry and Psychotherapy, Central Institute of Mental Health, Medical Faculty Mannheim/Heidelberg University, Mannheim, Germany
[b] Department of Child and Adolescent Psychiatry and Psychotherapy, Psychiatric Hospital, University of Zurich, Zurich, Switzerland
[c] Center for Integrative Human Physiology, University of Zurich, Zurich, Switzerland
[d] Neuroscience Center Zurich, University of Zurich and ETH Zurich, Zurich, Switzerland

ARTICLE INFO

Keywords:
Attention-deficit/hyperactivity disorder (ADHD)
Event-related potentials (ERPs)
Electroencephalography (EEG)
Meta-analysis
Cognitive modulation

ABSTRACT

The current meta-analysis summarizes relevant literature on earlier (P100, N100, P200, N200, ERN/Ne) versus later (P300, Pe, CNV) cognitive Event-Related Potential (ERP) differences between children, adolescents, and adults with Attention-Deficit/Hyperactivity Disorder (ADHD) and without ADHD (non-ADHD). Furthermore, the heterogeneity in previous research is addressed by analyzing potentially relevant demographic and methodological moderators (age group, IQ, medication, comorbidity, task, cognitive function, modality, inter-stimulus-interval, number of electrodes). Via database search 52 relevant articles were identified including $n = 1576$ ADHD and $n = 1794$ non-ADHD. Using multilevel-models, pooled effect sizes were calculated. For earlier components, individuals with ADHD showed shorter Go-P100-latencies than non-ADHD. For later ERPs, individuals with ADHD showed smaller Cue-P300-amplitudes, longer Go-P300-latencies, smaller NoGo-P300-amplitudes, longer NoGo-P300-latencies, smaller CNV-amplitudes, and smaller Pe-amplitudes. The substantial heterogeneity identified for most of the ERP components could be explained by the demographic and methodological moderators of interest. This meta-analysis identified relevant moderate group differences ($-0.32 < d < -0.57$), mainly regarding later cognitive ERPs. Nevertheless, results are characterized by substantial heterogeneity and the moderate effect sizes ($d < 0.6$) limit the use for clinical application.

1. Introduction

Attention-deficit/hyperactivity disorder (ADHD) is one of the most prevalent neurodevelopmental disorders characterized by core symptoms of age-inappropriate levels of inattention, hyperactivity, and impulsivity (Biederman and Faraone, 2005; Taylor et al., 2004). With a prevalence of approximately 5 % (Polanczyk et al., 2015) and a persistence rate of 30–40 % into adulthood (Faraone et al., 2006), it is considered as a major public health problem due to the significant social and educational disadvantage of the affected patients (Lesesne et al., 2000; Swanson et al., 1998). Several findings have already emphasized the biological underpinnings of ADHD (Thome et al., 2012). The study of biological markers in individuals with ADHD represents an important path towards understanding the clinically and etiologically heterogeneous nature of this neurodevelopmental disorder and its therapeutic outcomes (Faraone et al., 2014). While no single reliable biomarker for the diagnosis of ADHD exists to date, some promising candidate brain-based biomarkers have been discussed (Gamma and Kara, 2016). For instance, Event-Related Potentials (ERPs) during response inhibition and response control have been widely examined in ADHD (Gamma and Kara, 2016; Johnstone et al., 2013; Thome et al., 2012; see Szuromi et al., 2011 for a quantitative review on adult ADHD P300-differences).

1.1. Previous findings on event-related potentials in ADHD

Cognitive ERPs represent stimulus-locked time epochs in the electroencephalogramm (EEG). They offer a unique window into the brain and represent promising tools for exploring the biological basis of cognitive functioning in ADHD due to their ease of administration, their functional relevance, and their high time-resolution (Lenartowicz and Loo, 2014). As cognitive ERPs are defined by the time they occur after

* Corresponding author at: Department of Child and Adolescent Psychiatry and Psychotherapy, Central Institute of Mental Health, J5, 68159 Mannheim, Germany.
E-mail address: anna.kaiser@zi-mannheim.de (A. Kaiser).

https://doi.org/10.1016/j.neubiorev.2020.01.019
Received 12 June 2019; Received in revised form 15 January 2020; Accepted 18 January 2020
Available online 25 January 2020

Table 1
Overview of ERP components and their mental processing correlates.

ERP component	Mental processing correlates
P100[a]	Spatial attention; gating to stimulus location (Luck et al., 2000)
N100[b]	Spatial attention; orienting response; matching processes with previously experiences stimuli; processing of unexpected stimuli (Luck et al., 1990; Sur and Sinha, 2009)
P200[a]	Attention to/processing of visual stimuli; sensation-seeking (Sur and Sinha, 2009)
N200[b]	Processing of deviant stimuli; classification of stimulus (Sur and Sinha, 2009)
P300[a]	Stimulus processing & evaluation of task-relevance (Cortese, 2012); updating of working memory, event categorization, attentional resource allocation, and attentional reorientation (Polich, 2007)
CNV[b]	Stimulus expectation; motor and non-motor preprocessing after cue stimulus (Walter et al., 1964)
ERN/Ne[b]	Error detection; error correction (Coles and Rugg, 1995)
Pe[a]	Error processing (Nieuwenhuis et al., 2001)

[a] Positive wave/deflection.
[b] Negative wave/deflection.

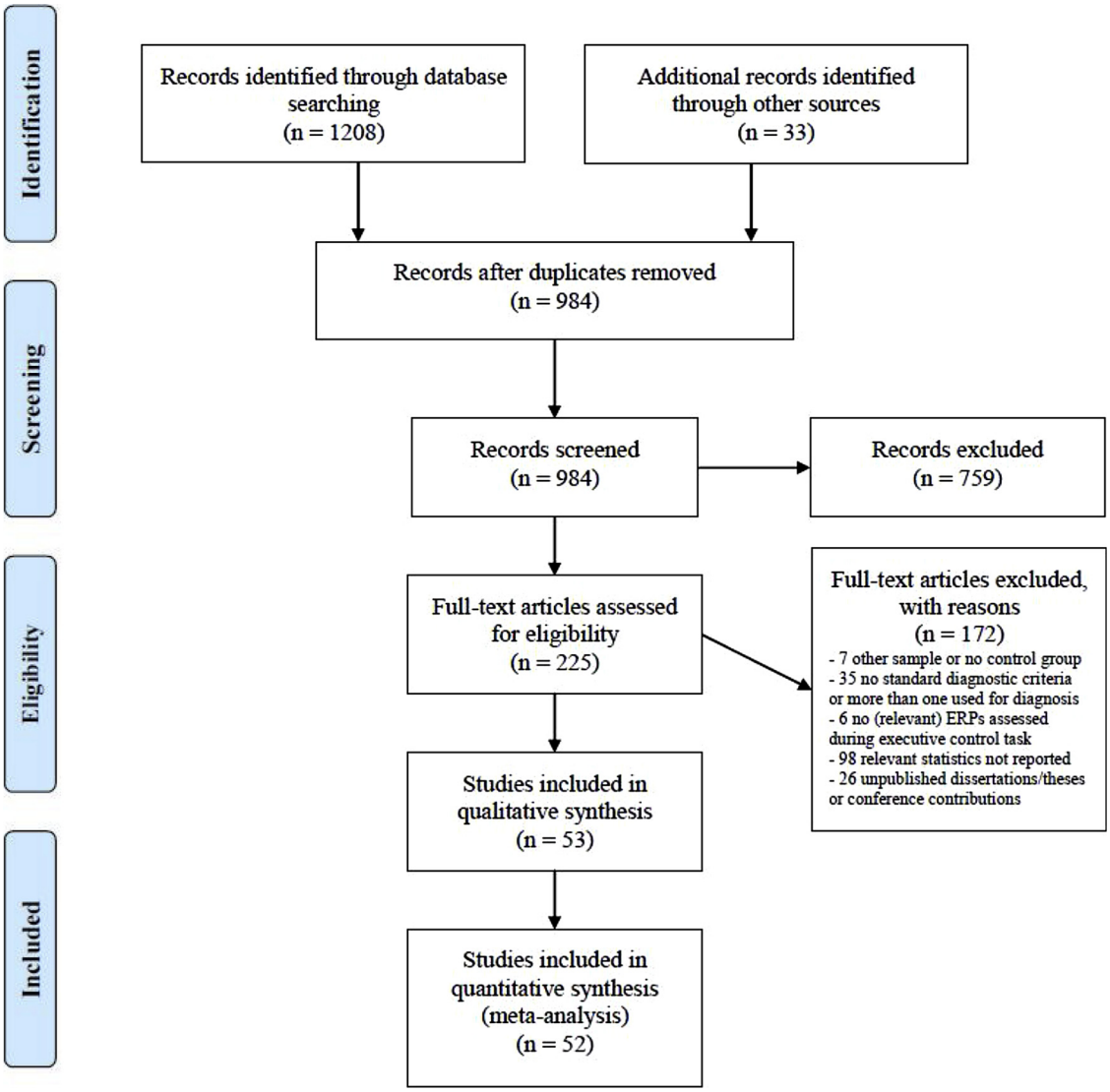

Fig. 1. Flow chart displaying the literature selection process according to PRISMA guidelines (Moher et al., 2009).

stimulus presentation in the task-related EEG, they can be divided into earlier (P100, N100, P200, N200, ERN/Ne) and later (P300, CNV, Pe) components reflecting the time course of task-related neural information-processing. For the current meta-analysis and in line with cognitive models of ADHD (e.g. Kofler et al., 2019), cognitive ERPs including sensory components with prominent cognitive modulation are in the focus of interest. Very early components reflecting mainly sensory processing, such as brain stem potentials or the P50 indexing sensory gating (e.g. Micoulaud-Franchi et al., 2015), were excluded. We also excluded the Mismatch Negativity (MMN) component that specifically assesses the integrity of automatic auditory-sensory memory and involuntary attentional switches outside the focus cognitive tasks. This component had been analyzed in a previous meta-analysis in children with ADHD (Cheng et al., 2016), suggesting a reduced MMN amplitude

Table 2
Description of included trials: Demographic information (across all ERP components).

	ADHD	Non-ADHD	*t*	df	*p*
N	1576	1794	–	–	–
$n_{children}$	840	742			
$n_{adolescents}$	275	542			
n_{adults}	461	510			
Age (years), *M* (*SD*)	15.52 (8.53)	15.45 (8.16)	0.04	104	0.96
Male (%), *M* (*SD*)	82.26 (17.12)	74.90 (20.90)	1.91	92	0.06
IQ, *M* (*SD*)	103.06 (7.10)	110.14 (6.37)	−4.23	63	< .0001

Note. Results for Welch-two-sample *t*-test (between-group comparison).

compared to children without ADHD. For a description of relevant ERP components addressed within the current analysis and their neuropsychological equivalent reflecting cognitive activation or modulation, see Table 1.

Several studies have documented robust neurophysiological differences between individuals with ADHD and individuals without ADHD (non-ADHD), especially for later ERPs, including lower NoGo-P300-amplitudes in individuals with ADHD over central regions during auditory and visual response-control tasks compared to non-ADHD children and adolescents, as well as reduced CNV-amplitudes in ADHD (e.g. reviewed in Barry et al., 2003). Regarding earlier ERPs during executive-control tasks, results are less consistent and more depending on potential influence variables: while several studies report on abnormalities of the N200-component (e.g. Albrecht et al., 2005; Pliszka et al., 2000; Tamayo-Orrego et al., 2015), others indicate that these only occur under specific task-conditions (e.g. Yong-Liang et al., 2000).

Extensive research has examined ERP differences between individuals with ADHD and individuals without ADHD, but until now there is no quantitative summary of previous literature systematically analyzing ERPs as possible markers of ADHD across the lifespan capitalizing on the high time resolution of ERPs by specifically taking into account differences between effects on earlier and later cognitive ERPs.

1.2. Potential sources of heterogeneity

The partly inconsistent findings described above might reflect the substantial heterogeneity of patient samples with ADHD (Lenartowicz and Loo, 2014): individual characteristics, such as age, IQ, medication status, symptom severity or the presence of comorbid disorders might influence neurophysiological processing (Bresnahan et al., 1999; Loo et al., 2013). Furthermore, methodological variations between the studies might contribute to the heterogeneity in previous findings. These include the specific task used to assess cognitive functioning, task-specific variations, such as the modality of stimulus presentation or the Inter-Stimulus-Interval (ISI) as well as technical, EEG-related between-study differences, such as the number of electrodes used to assess neurophysiological processing (e.g. Yong-Liang et al., 2000). To clarify the impact of demographic and methodological between-study differences, a systematic quantitative analysis on these potentially relevant moderator variables is urgently needed.

Based on previous qualitative and quantitative reviews (Gamma and Kara, 2016; Johnstone et al., 2013; Szuromi et al., 2011; Thome et al., 2012), a meta-analysis was conducted summarizing relevant literature on ERP differences in children, adolescents, and adults with ADHD compared to individuals without ADHD. The focus was on identifying group-level differences regarding earlier (P100, N100, P200, N200, ERN/Ne) versus later (P300, CNV, Pe) cognitive ERP components (amplitudes and latencies) during inhibitory control, attention, working memory, and performance monitoring using a quantitative approach. The main aim is to clarify when the most robust neurophysiological deviations occur in individuals with ADHD in the time course of cognitive processing covered by task-related ERPs. Generally, we assume smaller ERP components and longer ERP latencies in individuals with ADHD when compared to individuals without ADHD reflecting inefficient cognitive modulation in neuropsychological processing, especially during later processing-stages. Furthermore, the current work aimed at addressing the heterogeneity found in previous research by defining (based on previous studies) and analyzing (partly in an explorative way) potentially relevant demographic (age group, IQ, medication, comorbidities) and methodological (task, cognitive function assessed by task, modality of stimulus presentation, inter-stimulus-interval, number of electrodes used for analysis) moderators.

Table 3
Overall mean estimated true effect sizes for random-effects models/multilevel linear models.

	Amplitude				Latency			
ERP component	*k*	*d*	[*95% CI*]	Q_W (*df*, *p*)	*k*	*d*	[*95% CI*]	Q_W (*df*, *p*)
Cue trials								
P100	$k \leq 1$	*n.a.*	*n.a.*	*n.a.*	$k \leq 1$	*n.a.*	*n.a.*	*n.a.*
N100	$k \leq 1$	*n.a.*	*n.a.*	*n.a.*	$k \leq 1$	*n.a.*	*n.a.*	*n.a.*
P200	$k \leq 1$	*n.a.*	*n.a.*	*n.a.*	$k \leq 1$	*n.a.*	*n.a.*	*n.a.*
N200	$k \leq 1$	*n.a.*	*n.a.*	*n.a.*	$k \leq 1$	*n.a.*	*n.a.*	*n.a.*
P300	18	−0.56***	[-0.82 – (-0.30)]	22.04 (17, .18)	2	−0.35	[-0.80 – 0.10]	2.96 (1, .09)
Go trials								
P100	10	0.41	[-0.69 – 1.50]	61.80 (9, < .0001)	10	−0.33**	[-0.53 – (-0.13)]	8.28 (9, .51)
N100	14	−0.41	[-0.94 – 0.12]	68.41 (13, < .0001)	13	−0.03	[-0.40 – 0.34]	31.16 (12, .002)
P200	16	0.49	[-0.24 – 1.23]	95.68 (15, < .0001)	15	0.01	[-0.83 – 0.86]	106.20 (14, < .0001)
N200	48	0.14	[-0.08 – 0.35]	126.58 (47, < .0001)	31	−0.36	[-1.01 – 0.30]	140.36 (30, < .0001)
P300	76	−0.14	[-0.32 – 0.04]	216.96 (75, < .0001)	38	0.52*	[0.08 – 0.96]	201.33 (37, < .0001)
NoGo trials								
P100	2	−0.19	[-0.58 – 0.19]	0.01 (1, .93)	3	−0.13	[-0.48 – 0.22]	0.35 (2, .84)
N100	5	−0.11	[-0.38 – 0.17]	6.54 (4, .16)	6	0.04	[-0.22 – 0.30]	5.26 (5, .39)
P200	4	0.03	[-0.32 – 0.37]	7.63 (3, 0.05)	5	0.05	[-0.67 – 0.77]	10.65 (4, .03)
N200	16	0.08	[-0.19 – 0.36]	34.76 (15, .00)	5	−0.59	[-2.49 – 1.32]	38.52 (4, < .0001)
P300	37	−0.57***	[-0.90 – (-0.24)]	95.73 (36, < .0001)	9	0.35**	[0.11 – 0.58]	16.85 (8, .03)
CNV	15	0.32*	[0.03 – 0.61]	45.99 (14, < .0001)	$k \leq 1$	*n.a.*	*n.a.*	*n.a.*
ERN/Ne	23	0.21	[-0.06 – 0.47]	69.52 (22, < .0001)	12	0.04	[-0.40 – 0.48]	26.02 (11, .01)
Pe	23	−0.39**	[-0.64 – (-0.13)]	58.33 (22, < .0001)	8	−0.01	[-0.40 – 0.39]	7.37 (7, .39)

Note. *** $p < .001$, ** $p < .01$, * $p < .05$, ° $p < .1$. *n.a. not available.*

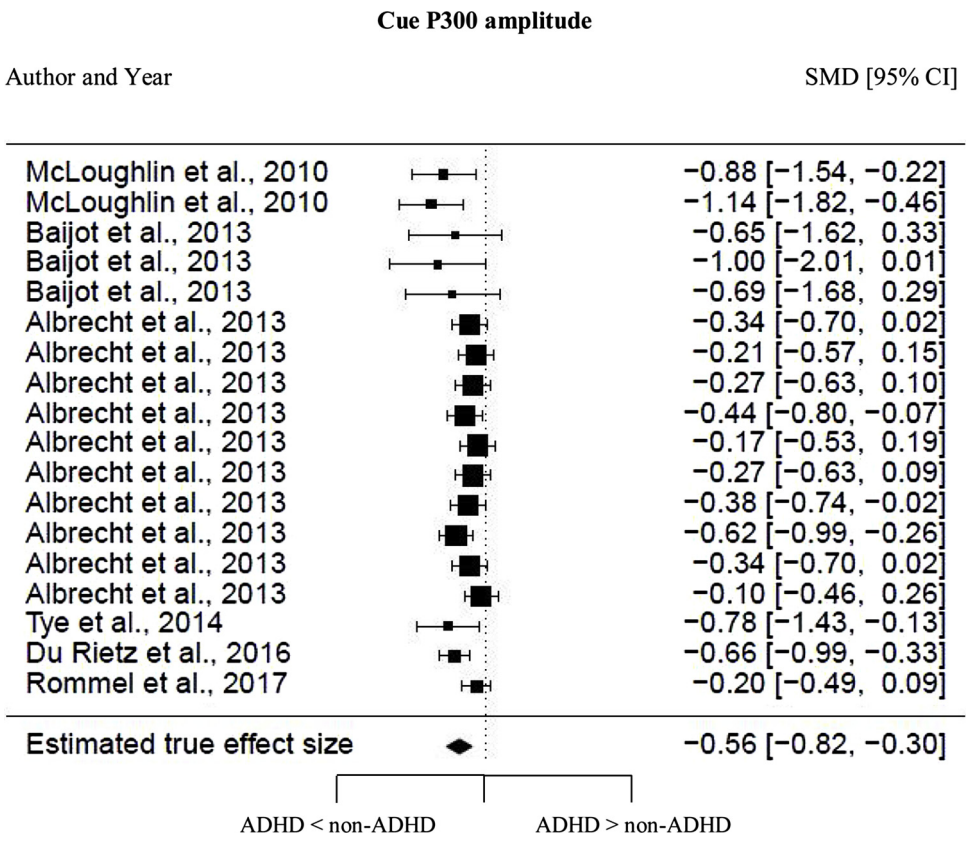

Fig. 2. Cumulative forest plot displaying meta-analytical results obtained from fitting multilevel models to Cue P300 amplitude data and addressing multilevel structure. *Note*. Multiple listings of the same study reflect different electrode localizations.

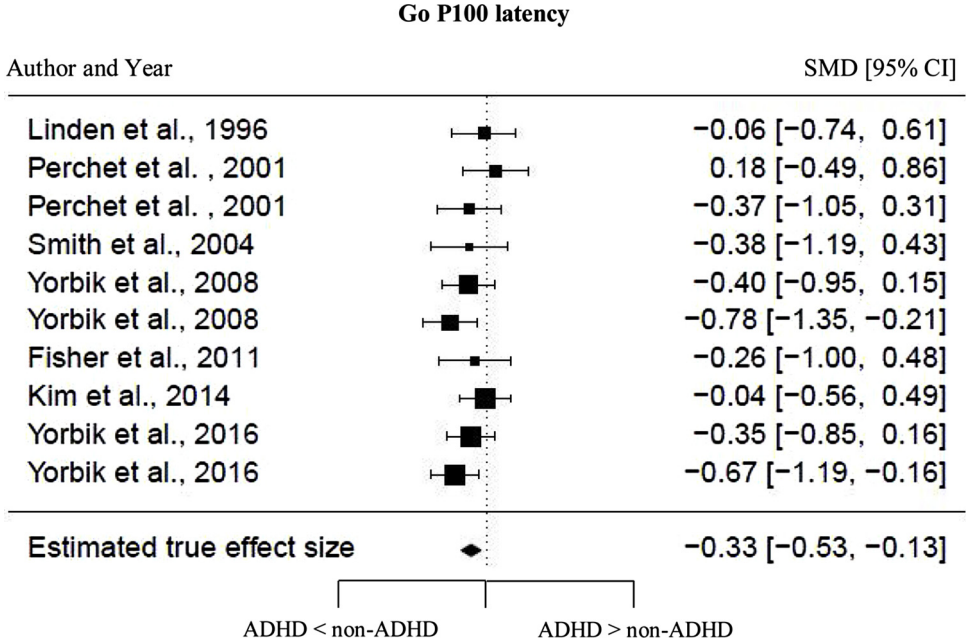

Fig. 3. Cumulative forest plot displaying meta-analytical results obtained from fitting multilevel models to Go P100 latency data and addressing multilevel structure. *Note*. Multiple listings of the same study reflect different electrode localizations.

2. Methods

2.1. Literature search and selection criteria

The current meta-analysis was registered on PROSPERO (http://www.crd.york.ac.uk/PROSPERO/display_record.php?ID=CRD42018098992).

The literature search was performed in line with the PRISMA-Statement (Moher et al., 2009), incorporating two different search strategies: An initial search was performed using the databases MEDLINE (via PubMed), PsychINFO, PsychARTICLES, Cochrane Central, and Clinical Trials. The subsequent keywords were entered: *ADHD*

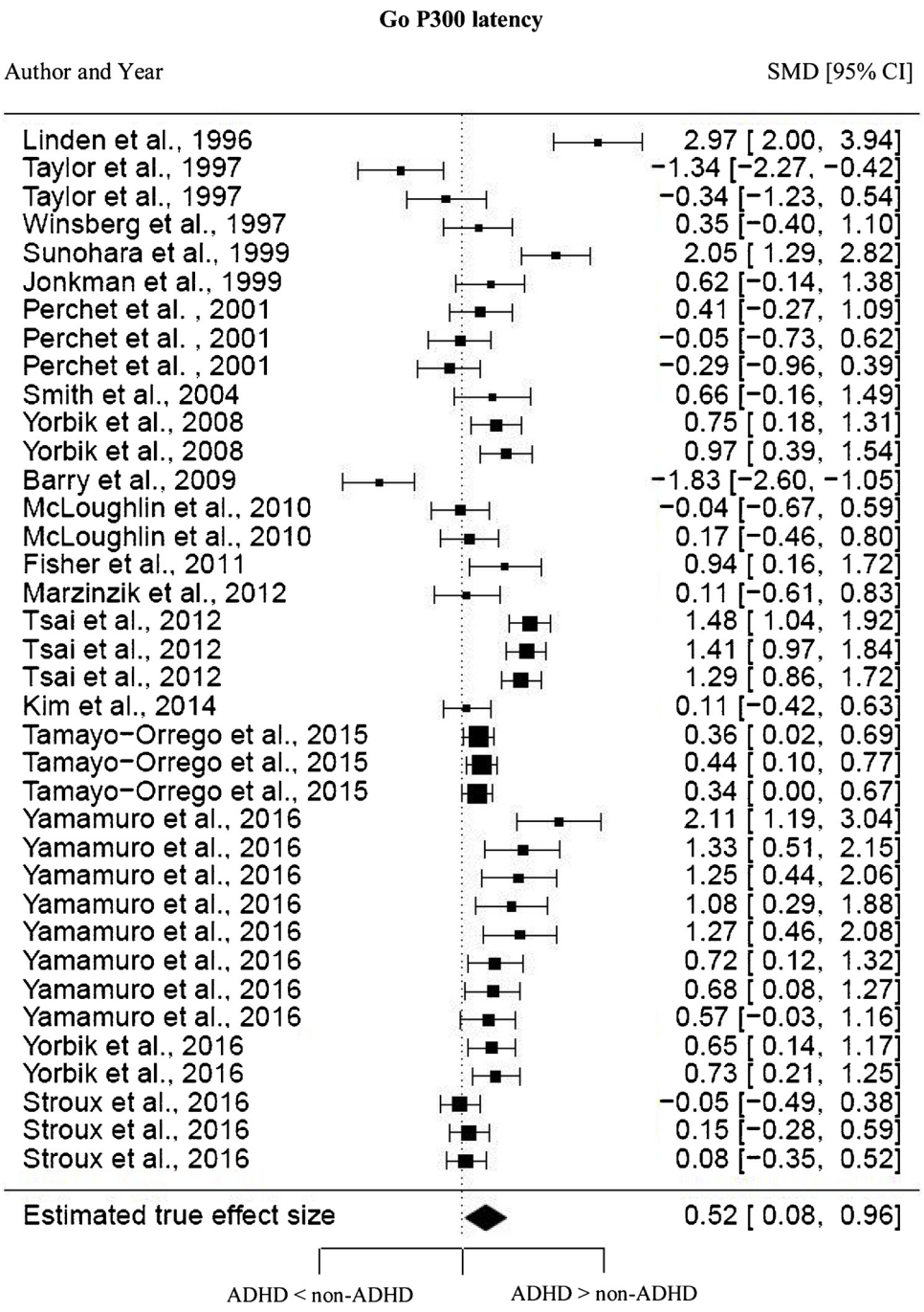

Fig. 4. Cumulative forest plot displaying meta-analytical results obtained from fitting multilevel models to Go P300 latency data and addressing multilevel structure. *Note*. Multiple listings of the same study reflect different age groups (Taylor et al., 1997) or electrode localizations.

(separately) combined with *EEG* or *ERP* (for more detailed information, see Appendix A in Supplementary material). The literature search was started in January 2018 and originally finished in April 2018. An update of the literature search was done in April 2019, but no new studies were relevant for inclusion. Second, additional records were identified by reviewing the reference lists of the papers included via the database search.

All studies identified were screened and assessed for eligibility according to the following inclusion/exclusion criteria:

(a) Reporting of quantitative data to compare ERP-markers of cognitive modulation between children, adolescents, and adults with and without ADHD.
(b) Administration of an EEG while participants engage in tasks involving inhibitory control, (selective) attention, working memory, and error monitoring to assess relevant ERPs.
(c) Examination of a group of individuals without ADHD compared to children, adolescents, and/or adults with ADHD.
(d) Formal diagnosis of ADHD according to only one of the following criteria: DSM-III-R, DSM-IV, DSM-IV-TR, DSM-V, or ICD-10 (refers to ADHD as hyperkinetic disorder; HKD).
(e) Study in one of the following languages: English, German, French, or Spanish.
(f) No case studies or review articles.
(g) Published study between January 1987 (publication year of DSM-III-R) and April 2018.

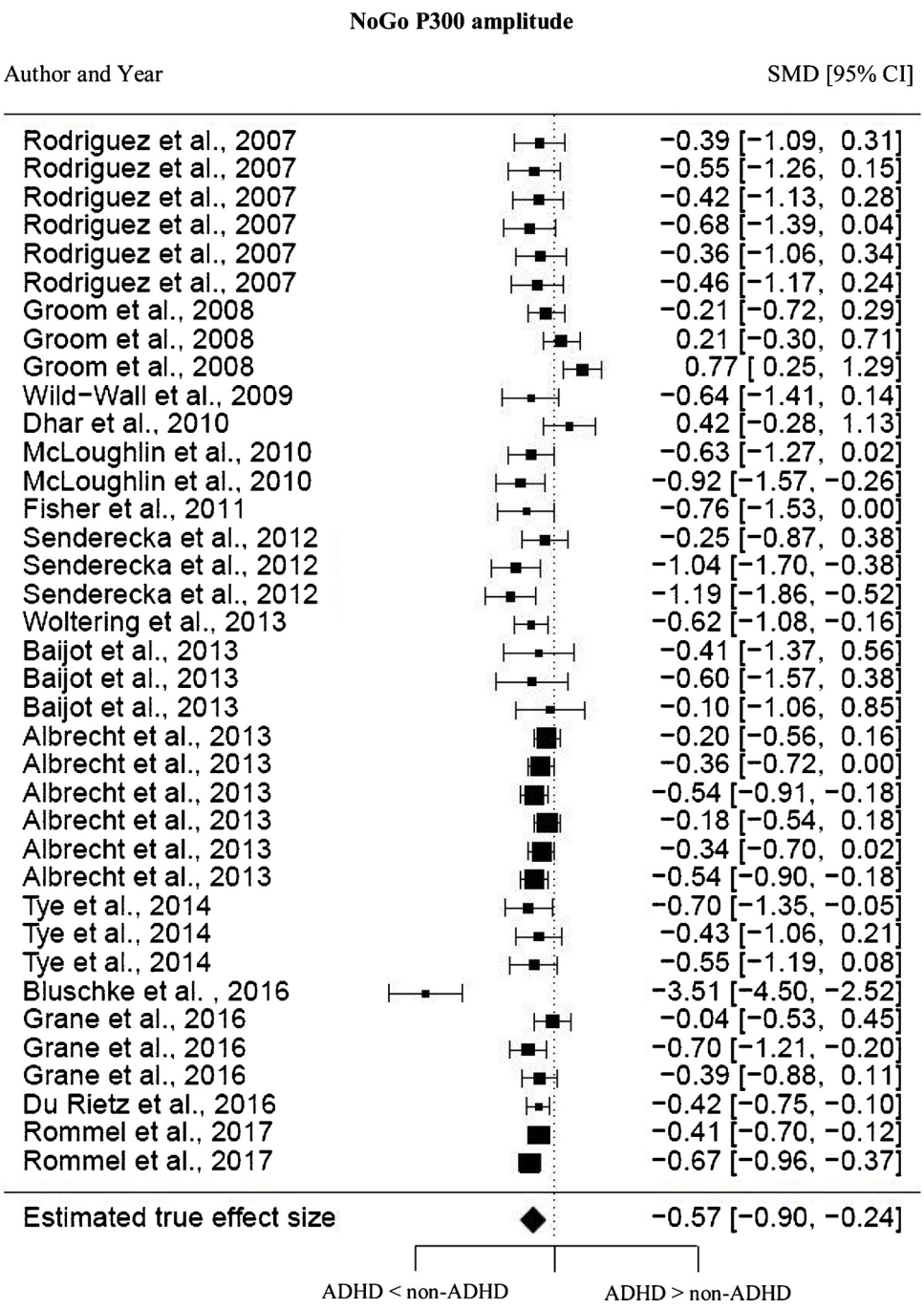

Fig. 5. Cumulative forest plot displaying meta-analytical results obtained from fitting multilevel models to NoGo P300 amplitude data and addressing multilevel structure. *Note*. Multiple listings of the same study reflect different ADHD subtypes (Rodriguez et al., 2007) or electrode localizations.

(h) Sufficient information to calculate the effect size.

A total of 984 potentially relevant studies were identified.[1] Fig. 1 provides an overview of the search process and the number of records included through each of the before mentioned search strategies. Finally, the literature search resulted in 52 studies for inclusion (Tamayo-Orrego et al., 2015)[2]. An asterisk in the reference list marks the included articles.

2.2. Data coding

A coding sheet was implemented to record all relevant variables (see Appendix B in Supplementary material). The relevant information was extracted from the articles and coded by the first and second author, independently from each other. Disagreement (< 5 %) was resolved in discussion.

The data sheets including the coded information used for subsequent analyses can be found in Appendix C in Supplementary material.

[1] After de-duplication

[2] One further study had to be excluded as the type of ERP indices reported could not be integrated into quantitative analyses.

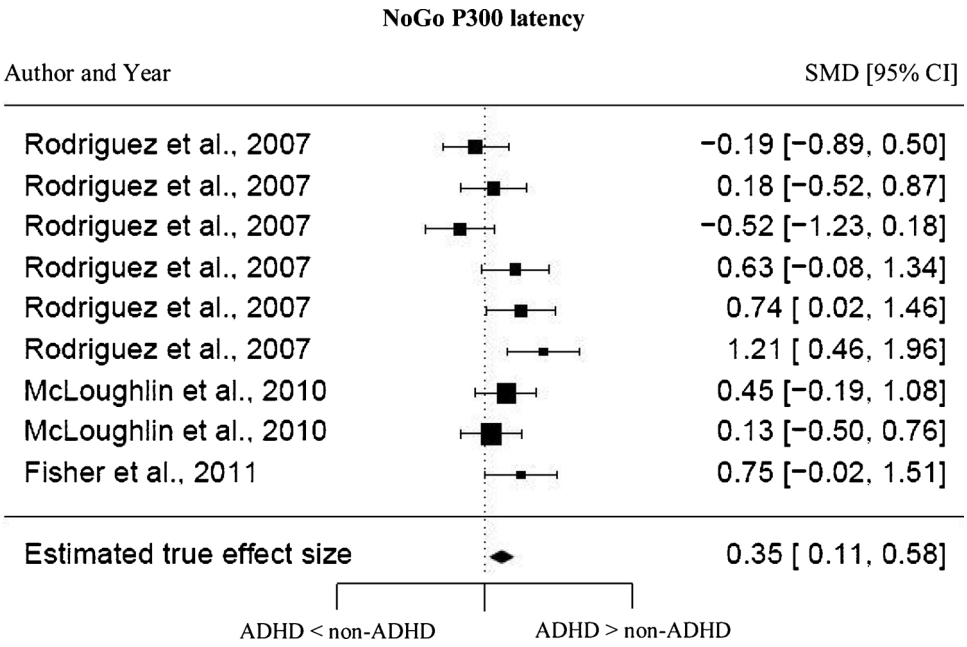

Fig. 6. Cumulative forest plot displaying meta-analytical results obtained from fitting multilevel models to NoGo P300 latency data and addressing multilevel structure. *Note*. Multiple listings of the same study reflect different ADHD subtypes (Rodriguez et al., 2007) or electrode localizations.

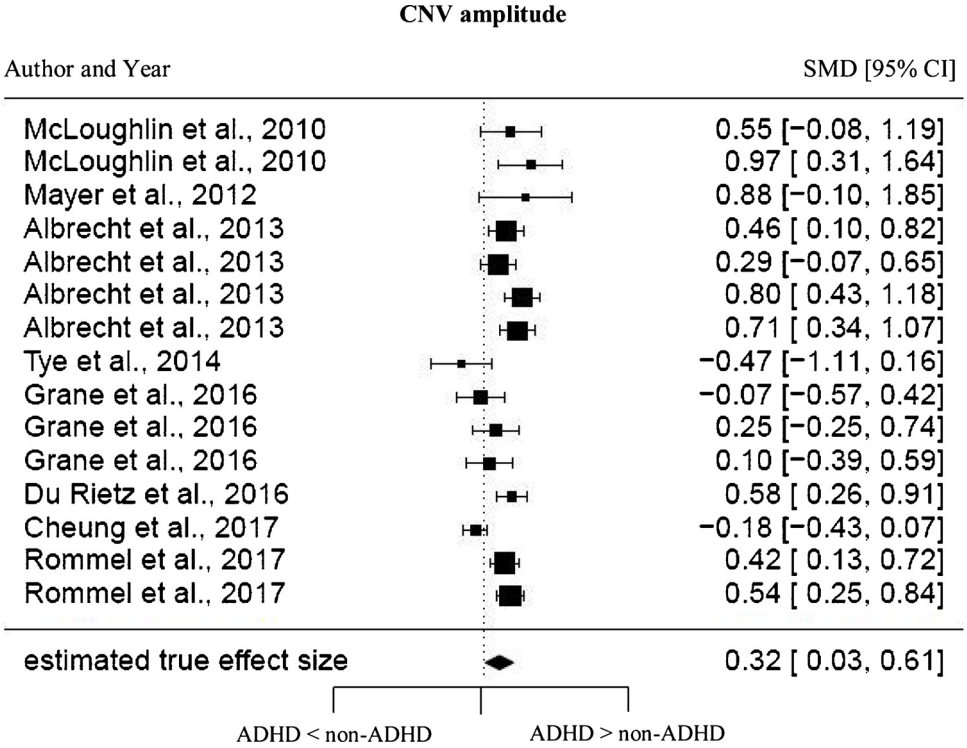

Fig. 7. Cumulative forest plot displaying meta-analytical results obtained from fitting multilevel models to CNV amplitude data and addressing multilevel structure. *Note*. Multiple listings of the same study reflect different electrode localizations.

2.3. Statistical analyses

For all analyses conducted, the *metafor* package (Viechtbauer, 2010; metafor Version 2.0.0, released on 22/06/2017) for R (R Development Core Team, 2018; R Version 3.5.1.) was used.

The standardized mean difference (*d*) in ERP amplitudes[3] and latencies between individuals with and without ADHD was computed as the relevant effect size measure (ADHD minus non-ADHD; Hedges and Olkin, 1985). Effect sizes were not recoded to have all expected effects in the same direction. For amplitudes of positive ERPs, negative effect sizes indicate smaller amplitudes in the ADHD group compared to non-ADHD. For amplitudes of negative ERPs, positive effect sizes indicate

[3] Group mean amplitudes (of individual peak latencies, peak amplitudes or mean amplitudes) are included as dependent variables of interest, as they are commonly reported.

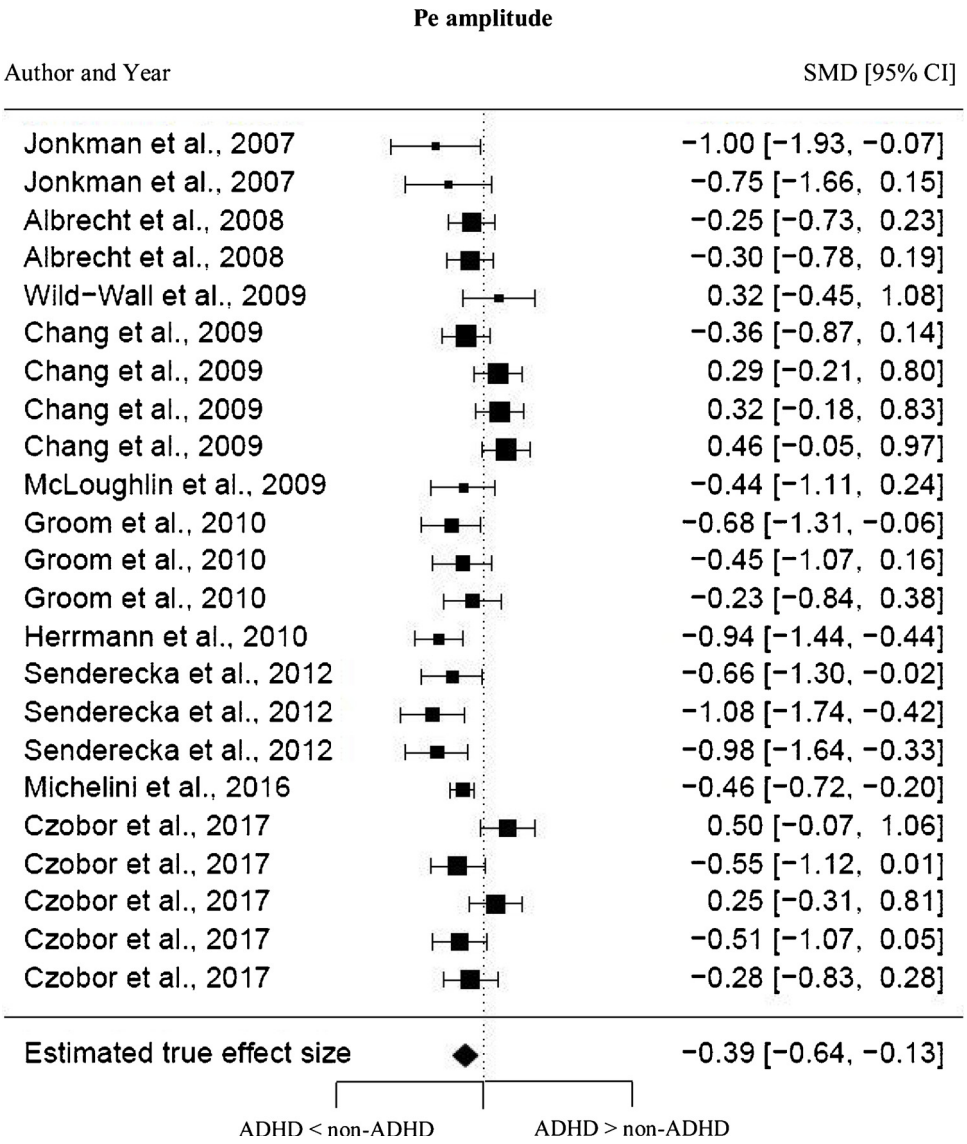

Fig. 8. Cumulative forest plot displaying meta-analytical results obtained from fitting multilevel models to Pe amplitude data and addressing multilevel structure. *Note*. Multiple listings of the same study reflect different electrode localizations.

smaller amplitudes in the ADHD group. For ERP latencies, positive mean effect sizes are associated with longer latencies in the ADHD group compared to non-ADHD. All effect sizes were calculated using exact statistics reported in the included studies. For each ERP component, a mean effect size was computed for the difference in amplitude and latency. Furthermore, effect sizes were calculated separately for each condition (Cue vs. Go vs. NoGo[4]) for the following ERP components: N100, P100, N200, P200, P300.

Multilevel models based on random-effects assumptions were fitted to the data to estimate the *true* mean effect sizes. Random-effects models were chosen because they allow for unconditional inferences above the specific study implementations (Borenstein et al., 2010). Multilevel models were implemented to address dependencies due to a multilevel structure in the data (more than one ES per study in same analysis e.g. due to more than one age group or neural activity on more than one electrode location assessed; Viechtbauer, 2010). For the estimation of the mean effect sizes, studies were weighted using the heteroscedastic sampling variance. To explore moderator effects, mixed-effects models were fitted to the data.

As an indicator of heterogeneity, the chi-square statistic Q (Cochrane`s Q-test; Hedges and Olkin, 1985) was calculated. The Q_W statistic obtained in the moderator analyses represents the residual heterogeneity after taking into account a moderator effect. The Q_B statistic refers to the test of a specific moderator. For estimating the amount of heterogeneity in the effect size distribution, the Restricted Maximum Likelihood (REML) estimator was used.

Furthermore, sensitivity analyses were conducted to test for the robustness of effects. To address the potential presence of publication bias, trim-and-fill analyses were calculated.

3. Results

3.1. Study characteristics

Characteristics of the included studies can be found in Table S1 (Appendix D in Supplementary material). A summary of demographic

[4] Cue, Go, and NoGo represent different task conditions. Cue – Cue stimulus presented to signal upcoming task, typically before target or NoGo stimulus. Go – Target stimulus presented that requires response (e.g. motor). NoGo – Stimulus presented that requires to inhibit prepared or prepotent response (e.g. motor).

Table 4a
Summary of meta-analytic findings for **amplitude** moderator analyses (mixed-effects models fitted) – P100, N100, P200.

	Cue				Go				NoGo			
Amplitude	k	Q_B(df, p)	Q_W(df, p)	Comparison	k	Q_B(df, p)	Q_W(df, p)	Comparison	k	Q_B(df, p)	Q_W(df, p)	Comparison
P100												
age	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*
IQ	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*
medication	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*
comorbidity	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*
task	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*
cogn. function	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*
modality	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*
ISI	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*
electrodes	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*
N100												
age	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*
IQ	*n.a.*	*n.a.*	*n.a.*	*n.a.*	8	5.48 (1,.02)	30.67 (6, < .0001)	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*
medication	*n.a.*	*n.a.*	*n.a.*	*n.a.*	14	34.02 (3, < .0001)	28.67 (11,.003)	2 < 3 < 1*** (neg)	*n.a.*	*n.a.*	*n.a.*	*n.a.*
comorbidity	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*
task	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*
cogn. function	*n.a.*	*n.a.*	*n.a.*	*n.a.*	14	6.42 (2, .04)	59.13 (12, < .0001)	2 < 1* (neg)	*n.a.*	*n.a.*	*n.a.*	*n.a.*
modality	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*
ISI	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*
electrodes	*n.a.*	*n.a.*	*n.a.*	*n.a.*	14	7.57 (1, .006)	54.05 (12, < .0001)	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*
P200											.	
age	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*
IQ	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*
medication	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*
comorbidity	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*
task	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*
cogn. function	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*
modality	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*
ISI	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*
electrodes	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*

Note. n.a. not available. *n.s.* not significant. Pos – positive ES. Neg – negative ES. Medication moderator: 1 – medicated; 2 – not medicated; 3 – washout period of 24 h; 4 – washout period of 48 h. Comorbidity moderator: 1 – yes, comorbid disorder present; 2 – no, no comorbid disorder present. Cognitive function moderator: 1 – Inhibition; 2 – Attention; 3 – Working memory; 4 – Error processing. Modality moderator: 1 – visual; 2 – auditory; 3 – multimodal.

study characteristics across all ERP components (P100, N100, P200, N200, P300, CNV, ERN/Ne, Pe), conditions (Cue versus Go versus NoGo), and dependent variables (amplitude versus latency) is presented in Table 2 (a summary of methodological characteristics can be found in Table S2; Appendix E in Supplementary material. Table S3 (Appendix E in Supplementary material) displays the relevant demographic characteristics separately for each ERP component, condition, and each dependent variable.

3.2. Overall mean effects

Overall mean estimated effect sizes obtained from fitting multilevel models and corresponding heterogeneity estimates are presented in Table 3.

3.2.1. Cue condition

For the P300-amplitude, the analysis reveals a significant negative mean estimated effect size (d=-0.56 [-0.82 – (-0.30)]), indicating a smaller Cue-P300-amplitude in ADHD compared to non-ADHD. The P300-latency analysis resulted in a non-significant negative mean effect size. Fig. 2 displays the forest plot for the Cue-P300-amplitude[5] .

3.2.2. Go condition

Significant mean estimated effect sizes were obtained for the P100-latency (d=-0.33 [-0.53 – (-0.13)]), and the P300-latency (d = 0.52 [0.08 – 0.96]), indicating shorter Go-P100-latencies, and longer Go-P300-latencies in ADHD compared to non-ADHD. Regarding other ERP components, no significant group differences emerged. Figs. 3 and 4 show the forest plots for significant results obtained for the Go condition.

3.2.3. NoGo condition

The P300-amplitude (d= -0.57 [-0.90 – (-0.24)]) and the P300-latency components (d = 0.35 [0.11 – 0.58]) resulted in significant mean group differences. For the P300-amplitude, the results indicate that individuals with ADHD overall present with smaller P300-amplitudes compared to non-ADHD. The P300-latency results reveal a significantly higher mean latency in ADHD compared to non-ADHD. For other NoGo-ERP components, the results did not reach significance. The forest plots for the significant NoGo condition results can be found in Figs. 5 and 6.

3.2.4. CNV

A significant mean estimated effect size emerged for the CNV-amplitude (d = 0.32 [0.03 – 0.61]), indicating smaller CNV-amplitudes in ADHD compared to non-ADHD. Fig. 7 shows the forest plot for the CNV-amplitude component.

3.2.5. ERN/Ne, Pe

A significant mean group difference emerged for the Pe-amplitude (d=-0.39 [-0.64 – (-0.13)]), indicating smaller Pe-amplitudes in ADHD compared to non-ADHD. No further significant results could be obtained. Fig. 8 shows the forest plot for the Pe-amplitude component.

The forest plots for all non-significant results can be found in Appendix F in Supplementary material. Furthermore, Appendix G in Supplementary material presents the funnel plots for all ERP

[5] Some studies provide more than one effect size for ERP analyses reflecting distinct demographic (e.g. more than one age group assessed) and methodological aspects (e.g. ERP assessed at several different electrode positions). See Appendix C for a detailed presentation of demographic and methodological detail.

Table 4b
Summary of meta-analytic findings for **amplitude** moderator analyses (mixed-effects models fitted) –N200, P300.

	Cue				Go				NoGo			
Amplitude	*k*	$Q_B(df, p)$	$Q_W(df, p)$	*Comparison*	*k*	$Q_B(df, p)$	$Q_W(df, p)$	*Comparison*	*k*	$Q_B(df, p)$	$Q_W(df, p)$	*Comparison*
N200												
age	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*
IQ	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*
medication	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*
comorbidity	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*
task	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*
cogn. function	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*
modality	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*
ISI	*n.a.*	*n.a.*	*n.a.*	*n.a.*	40	5.86 (1, .02)	60.19 (38, .01)	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*
electrodes	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*
P300												
age	18	19.62 (3,.001)	14.81 (15, 0.47)	Adolescents* < Children** < Adults** (neg)	*n.s.*	*n.s.*	*n.s.*	*n.a.*	37	12.84 (3, .005)	92.85 (34, < .0001)	Adolescents < Adults < Children** (neg)
IQ	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*
medication	18	14.04 (3, .003)	16.46 (15, 0.35)	2 < 4** < 1° (neg)	*n.s.*	*n.s.*	*n.s.*	*n.a.*	35	13.31 (4, .01)	80.40 (31, < .0001)	3 < 2 < 4 < 1** (neg)
comorbidity	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	25	41.17 (2, < .0001)	21.54 (23, < 0001)	1** < 2*** (neg)
task	18	17.11 (2, < .001)	21.74 (16, 0.15)	2*** > 1*** (neg)	*n.s.*	*n.s.*	*n.s.*	*n.a.*	37	10.77 (4, .03)	93.92 (33, < .0001)	1 < 2 < 13 < 3** (neg)
cogn. function	18	18.05 (2, .0001)	16.99 (16, 0.39)	2*** < 1 (neg)	*n.s.*	*n.s.*	*n.s.*	*n.a.*	37	11.12 (2, .004)	94.49 (35, < .0001)	2 < 1** (neg)
modality	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	37	9.57 (3, .02)	90.92 (34, < .0001)	1** < 2 < 3 (neg)
ISI	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*
electrodes	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*

Note. n.a. not available. *n.s.* not significant. Pos – positive ES. Neg – negative ES. Medication moderator: 1 – medicated; 2 – not medicated; 3 – washout period of 24 h; 4 – washout period of 48 h. Comorbidity moderator: 1 – yes, comorbid disorder present; 2 – no, no comorbid disorder present. Cognitive function moderator: 1 – Inhibition; 2 – Attention; 3 – Working memory; 4 – Error processing. Modality moderator: 1 – visual; 2 – auditory; 3 – multimodal.

Table 4c
Summary of meta-analytic findings for **amplitude** moderator analyses (mixed-effects models fitted) – CNV, ERN/Ne, Pe.

Amplitude	*k*	$Q_B(df, p)$	$Q_W(df, p)$	*Comparison*
CNV				
age	*n.s.*	*n.s.*	*n.s.*	*n.a.*
IQ	*n.s.*	*n.s.*	*n.s.*	*n.a.*
medication	*n.s.*	*n.s.*	*n.s.*	*n.a.*
comorbidity	9	46.14 (2, < .0001)	6.96 (7, .43)	2*** < 1 (pos)
task	15	9.85 (4, .04)	18.57 (11, .07)	1 (pos) < 7 (neg) < 3 (pos) < 2*
cogn. function	*n.s.*	*n.s.*	*n.s.*	*n.a.*
modality	15	5.57 (2, .06)	44.87 (13, < .0001)	1° < 2 (pos)
ISI	14	8.30 (1, .004)	17.18 (12, .14)	*n.a.*
electrodes	*n.s.*	*n.s.*	*n.s.*	*n.a.*
ERN/Ne				
age	*n.s.*	*n.s.*	*n.s.*	*n.a.*
IQ	*n.s.*	*n.s.*	*n.s.*	*n.a.*
medication	*n.s.*	*n.s.*	*n.s.*	*n.a.*
comorbidity	*n.s.*	*n.s.*	*n.s.*	*n.a.*
task	*n.s.*	*n.s.*	*n.s.*	*n.a.*
cogn. function	*n.s.*	*n.s.*	*n.s.*	*n.a.*
modality	*n.s.*	*n.s.*	*n.s.*	*n.a.*
ISI	*n.s.*	*n.s.*	*n.s.*	*n.a.*
electrodes	*n.s.*	*n.s.*	*n.s.*	*n.a.*
Pe				
age	23	10.14 (3, .02)	50.16 (20, .0002)	Adolescents < Adults° < Children** (neg)
IQ	*n.s.*	*n.s.*	*n.s.*	*n.a.*
medication	22	31.03 (4, < .0001)	29.01 (18, .05)	1 (pos) < 4** < 3** < 2* (neg)
comorbidity	19	6.34 (2, .04)	44.53 (17, .0003)	1 < 2* (neg)
task	23	8.26 (2, .02)	57.17 (21, < .0001)	13* < 3* (neg)
cogn. function	23	8.24 (2, .02)	53.45 (21, < .0001)	4* < 1 (neg)
modality	23	12.62 (2, .002)	47.73 (21, .0007)	1* < 3* (neg)
ISI	*n.s.*	*n.s.*	*n.s.*	*n.a.*
electrodes	*n.s.*	*n.s.*	*n.s.*	*n.a.*

Note. n.a. not available. *n.s.* not significant. Pos – positive ES. Neg – negative ES. Medication moderator: 1 – medicated; 2 – not medicated; 3 – washout period of 24 h; 4 – washout period of 48 h. Comorbidity moderator: 1 – yes, comorbid disorder present; 2 – no, no comorbid disorder present. Cognitive function moderator: 1 – Inhibition; 2 – Attention; 3 – Working memory; 4 – Error processing. Modality moderator: 1 – visual; 2 – auditory; 3 – multimodal.

components, for amplitude and latency, respectively.

3.3. Moderator effects

As suggested by the *Q*-statistics obtained in the overall analyses, there is substantial heterogeneity in the distribution of effect sizes. To explore this heterogeneity, moderator analyses were implemented. Due to a lack of reporting and many different scales used to assess ADHD symptom severity, no moderator analysis could be conducted on this potentially relevant influence variable. As can be seen from Tables 4a–4c and Tables 5a–5c, significant moderator results were identified for all moderator variables postulated. For all categorical moderators, subgroup-comparisons are presented within the tables.

3.3.1. Age

For age moderator analyses, larger mean effect sizes were identified in children compared to adolescents or adults for the NoGo-P300-amplitude ($Q_B(2) = 12.84$, $p = .005$), the Pe-amplitude ($Q_B(3) = 10.14$, $p = .02$), the Go-P100-latency ($Q_B(2) = 13.55$, $p = .001$), the Go-P300-latency ($Q_B(2) = 10.49$, $p = .005$), and the NoGo-N200-latency ($Q_B(2) = 22.07$, $p < .0001$). For the Cue-P300-amplitude component on the contrary, largest mean effect sizes were obtained in adults ($Q_B(3) = 19.62$, $p = .001$).

3.3.2. IQ

A (marginally) significant positive relationship emerged between IQ and the sizes of the effects for the Go-P200-latency ($Q_B(1) = 2.71$, $p = .10$), Go-N200-latency ($Q_B(1) = 3.29$, $p = .07$), Go-N100-amplitude ($Q_B(1) = 5.48$, $p = .02$), NoGo-P200-latency ($Q_B(1) = 7.27$, $p = .007$), Go-N100-latency ($Q_B(1) = 9.17$, $p = .003$), NoGo-N200-latency, ($Q_B(1) = 32.84$, $p < .0001$).

3.3.3. Medication status

A significant association between the medication status of the ADHD group and the mean size of the effect was obtained for the following components: NoGo-P300-latency ($Q_B(2) = 7.46$, $p = .02$), NoGo-P300-amplitude ($Q_B(4) = 13.31$, $p = .01$), Cue-P300-amplitude ($Q_B(4) = 14.04$, $p = .003$), Go-P100-latency ($Q_B(2) = 11.42$, $p = .003$), Go-N100-amplitude ($Q_B(3) = 34.02$, $p < .0001$), and Pe-amplitude ($Q_B(4) = 31.03$, $p < .0001$).

3.3.4. Comorbidity

For comorbidity, a significant influence on mean effect size was obtained for the Go-P300-latency ($Q_B(2) = 6.58$, $p = .04$), the Pe-amplitude ($Q_B(2) = 6.34$, $p = .04$), the Go-P100-latency ($Q_B(2) = 7.69$, $p = .02$), the NoGo-N200-latency ($Q_B(2) = 7.64$, $p = .02$), the NoGo-P300-amplitude ($Q_B(2) = 41.17$, $p < .0001$), and the CNV-amplitude ($Q_B(2) = 46.14$, $p < .0001$) component,

3.3.5. Task

A significant moderator effect for task was revealed for the following ERPs: NoGo-P300-latency ($Q_B(3) = 7.94$, $p = .05$), Go-N100-latency ($Q_B(4) = 10.26$, $p = .04$), CNV-amplitude ($Q_B(4) = 9.85$, $p = .04$), NoGo-P300-amplitude ($Q_B(4) = 10.77$, $p = .03$), Pe-amplitude ($Q_B(2) = 8.26$, $p = .02$), Go-P100-latency ($Q_B(5) = 17.36$, $p = .004$), and Cue-P300-amplitude ($Q_B(2) = 17.11$, $p < .001$), with largest effect sizes for the CPT, the CPT-Flanker version, the Go/NoGo, and the Oddball task,

3.3.6. Cognitive function

The cognitive function moderator analysis resulted in significant effects for the the Go-N100-amplitude ($Q_B(2) = 6.42$, $p = .04$), the NoGo-P300-latency ($Q_B(2) = 7.46$, $p = .02$), and the Pe-amplitude ($Q_B(2) = 8.24$, $p = .02$), the Go-P100-latency ($Q_B(3) = 12.68$, $p = .005$), the NoGo-P300-amplitude ($Q_B(2) = 11.12$, $p = .004$), Cue-P300-amplitude ($Q_B(2) = 18.05$, $p = .0001$), indicating especially large effect sizes for tasks assessing inhibition.

3.3.7. Modality

Regarding the modality of stimulus presentation, significant moderator effects were obtained for the following components: NoGo-P300-amplitude ($Q_B(9) = 9.57$, $p = .02$), NoGo-P300-latency ($Q_B(2) = 9.74$, $p = .008$), Pe-amplitude ($Q_B(2) = 12.62$, $p = .002$), Go-P100-latency ($Q_B(2) = 15.39$, $p = .001$), Go-P300-latency ($Q_B(3) = 15.84$, $p = .0001$), with large effect sizes for auditory stimuli compared to visual stimuli for the Go-P100-latency, the Go-P300-latency, theNoGo-P300-amplitude, and the NoGo-P300-latency.

3.3.8. ISI

A significant moderator effect of the ISI on mean effect size was revealed for the Go-N200-amplitude ($Q_B(1) = 5.86$, $p = .02$), the Pe-latency ($Q_B(1) = 6.83$, $p = .009$), the CNV-amplitude ($Q_B(1) = 8.30$, $p = .004$), and the Go-N100-latency ($Q_B(1) = 14.26$, $p = .0002$), all indicating a small positive relationship between the length of the inter-stimulus-interval in the task and the size of the mean group difference.

Table 5a
Summary of meta-analytic findings for **latency** moderator analyses (mixed-effects models fitted) – P100, N100, P200.

	Cue				Go				NoGo			
Latency	*k*	$Q_B(df, p)$	$Q_W(df, p)$	*Comparison*	*k*	$Q_B(df, p)$	$Q_W(df, p)$	*Comparison*	*k*	$Q_B(df, p)$	$Q_W(df, p)$	*Comparison*
P100		.										
age	*n.a.*	*n.a.*	*n.a.*	*n.a.*	10	13.55 (2, .001)	6.92 (8, 0.55)	Adults < Children*** (neg)	*n.s.*	*n.s.*	*n.s.*	*n.a.*
IQ	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*
medication	*n.a.*	*n.a.*	*n.a.*	*n.a.*	8	11.42 (2, .003)	5.70 (6, .46)	3 < 2** (neg)	*n.s.*	*n.s.*	*n.s.*	*n.a.*.
comorbidity	*n.a.*	*n.a.*	*n.a.*	*n.a.*	4	7.69 (2, .02)	1.13 (2, .57)	1 < 2** (neg)	*n.s.*	*n.s.*	*n.s.*	*n.a.*
task	*n.a.*	*n.a.*	*n.a.*	*n.a.*	10	17.36 (5, .004)	3.12 (5, .68)	11 < 5 < 10 < 3 < 6*** (neg)	*n.a.*	*n.a.*	*n.a.*	*n.a.*
cogn. function	*n.a.*	*n.a.*	*n.a.*	*n.a.*	10	12.68 (3, .005)	6.73 (7, .46)	3 < 1 < 2*** (neg)	*n.a.*	*n.a.*	*n.a.*	*n.a.*
modality	*n.a.*	*n.a.*	*n.a.*	*n.a.*	10	15.39 (2, .001)	5.09 (8, .75)	1 < 2*** (neg)	*n.s.*	*n.s.*	*n.s.*	*n.a.*
ISI	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*
electrodes	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*
N100												
age	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*
IQ	*n.a.*	*n.a.*	*n.a.*	*n.a.*	8	9.17 (1, .003)	7.90 (6,.25)	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*
medication	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*.
comorbidity	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*
task	*n.a.*	*n.a.*	*n.a.*	*n.a.*	13	10.26 (4, .04)	14.26 (9, .11)	5 < 1 < 3 (neg) < 6* (pos)	*n.s.*	*n.s.*	*n.s.*	*n.a.*
cogn. function	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*
modality	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	
ISI	*n.a.*	*n.a.*	*n.a.*	*n.a.*	9	14.26 (1, .0002)	7.40 (7, .39)	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*
electrodes	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*
P200												
age	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*.	*n.s.*	*n.s.*	*n.s.*	*n.a.*
IQ	*n.a.*	*n.a.*	*n.a.*	*n.a.*	12	2.71 (1, .10)	37.90 (10, < .0001)	*n.a.*	4	7.27 (1, .007)	3.14 (2, 0.21)	*n.a.*
medication	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*.	*n.s.*	*n.s.*	*n.s.*	*n.a.*.
comorbidity	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*
task	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*
cogn. function	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*
modality	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*
ISI	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*
electrodes	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	5	6.60 (1, .01)	4.06 (3, 0.26)	*n.a.*

Note. n.a. not available. *n.s.* not significant. Pos – positive ES. Neg – negative ES. Medication moderator: 1 – medicated; 2 – not medicated; 3 – washout period of 24 h; 4 – washout period of 48 h. Comorbidity moderator: 1 – yes, comorbid disorder present; 2 – no, no comorbid disorder present. Cognitive function moderator: 1 – Inhibition; 2 – Attention; 3 – Working memory; 4 – Error processing. Modality moderator: 1 – visual; 2 – auditory; 3 – multimodal.

3.3.9. Number of electrodes

For the NoGo-P200-latency ($Q_B(1) = 6.600$, $p = .01$), a significant positive moderator effect could be obtained, indicating larger effect sizes with a higher number of electrodes used for the EEG assessment. On the other hand, for the Go-N100-amplitude ($Q_B(1) = 7.57$, $p = .006$) a significant negative effect of the moderator was identified: a higher number of electrodes is associated with smaller effect sizes.

3.4. Sensitivity analyses

Two sensitivity analyses were conducted to compare meta-analytic results obtained (I) from analyses with and without outlying studies[6] and (II) when there was no separation of different conditions (Cue, Go, NoGo). Results can be found in Tables S4–S9[7] (Appendix E in Supplementary material). Notably, for the Go-P300-amplitude and the CNV-amplitude, an even larger negative effect size was obtained after excluding outlying studies (d = -0.18 [-0.34 – (-0.02)], d = 0.41 [0.16 – 0.67], respectively). Furthermore, significant between-group differences for the overall P300-amplitude (d = -0.25 [-0.43 – (-0.08)]) and latency analyses (d = 0.50 [0.09 – 0.91]) emerged when fitting multilevel-models across Cue, Go, and NoGo conditions.

3.5. Comparison between earlier and later ERPs

For a direct comparison between earlier and later ERPs, a moderator analysis was implemented including data for all ERPs per trial condition (Cue, Go, NoGo[8]). A significant moderator effect for earlier versus later ERP components was obtained for the amplitudes of Cue-ERPs ($Q_M(2) = 123.71$, $p < .0001$), the amplitudes and latencies of Go-ERPs ($Q_M(7) = 80.65$, $p < .0001$, and $Q_M(7) = 113.24$, $p < .0001$), and the amplitudes of NoGo-ERPs ($Q_M(7) = 66.03$, $p < .0001$), with significant effects for the following ERPs: Cue-P300-amplitude, CNV-amplitude, Go-N200-amplitude, Go-P100-amplitude, Pe-amplitude, Go-N100-latency, Go-P300-latency, and NoGo-P300-amplitude[9].

[6] To determine statistical outliers, plots of the externally standardized residuals and Cook´s distances provided within the R package were examined.

[7] 1 effect size excluded for: Go-P100-amplitude, N100-amplitude, P200-amplitude, P300 latency, NoGo-N100-amplitude, P300-amplitude, P100-latency. 2 effect sizes excluded for: Go-N200-latency, NoGo-N100-latency, and ERN-amplitude. 3 effect sizes excluded for Go-P300-amplitude.

[8] CNV data were included in the Cue-dataset, while ERN/Ne and Pe data were included in Go- and NoGo-datasets for comparison.

[9] Deviations in results (when compared to overall results, separately conducted for each ERP) are due to a larger number of studies.

Table 5b
Summary of meta-analytic findings for **latency** moderator analyses (mixed-effects models fitted) –N200, P300.

	Cue				Go				NoGo			
Latency	*k*	$Q_B(df, p)$	$Q_W(df, p)$	*Comparison*	*k*	$Q_B(df, p)$	$Q_W(df, p)$	*Comparison*	*k*	$Q_B(df, p)$	$Q_W(df, p)$	*Comparison*
N200												
age	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	5	22.07 (2, < .0001)	6.77 (3, .08)	Adults (pos) < Children*** (neg)
IQ	*n.a.*	*n.a.*	*n.a.*	*n.a.*	13	3.29 (1, .07)	14.92 (11, .19)	*n.a.*	3	32.84 (1, < .0001)	0.02 (1, 0.88)	*n.a.*
medication	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*.	*n.s.*	*n.s.*	*n.s.*	*n.a.*.
comorbidity	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	4	7.64 (2, .02)	1.00 (2, 0.61)	1 (neg) < 2** (pos)
task	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*
cogn. function	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*
modality	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*
ISI	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*
electrodes	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*
P300												
age	*n.a.*	*n.a.*	*n.a.*	*n.a.*	38	10.49 (2, .005)	156.35 (36, < .0001)	Adults (neg) < Children*** (pos)	*n.a.*	*n.a.*	*n.a.*	*n.a.*
IQ	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*
medication	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	9	7.46 (2, .02)	16.75 (7, .02)	4 < 3* (pos)
comorbidity	*n.a.*	*n.a.*	*n.a.*	*n.a.*	15	6.58 (2, .04)	35.71 (13, .0007)	2 < 1* (pos)	*n.a.*	*n.a.*	*n.a.*	*n.a.*
task	2	5.25 (2, .07)	0.00 (0, 1.00)	1 (positive) < 2* (negative)	*n.s.*	*n.s.*	*n.s.*	*n.a.*	9	7.94 (3, .05)	16.26 (6, .01)	2 < 3* < 1 (pos)
cogn. function	*n.a.*	*n.a.*	*n.a.*		*n.s.*	*n.s.*	*n.s.*	*n.a.*	9	7.46 (2, .02)	16.75 (7, .02)	2 < 1* (pos)
modality	*n.a.*	*n.a.*	*n.a.*	*n.a.*	38	15.84 (3, .0001)	113.69 (35, < .0001)	1 (pos) < 3 (neg) < 2*** (pos)	9	9.74(2, .008)	15.69 (7, .03)	1* < 2° (pos)
ISI	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*
electrodes	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*	*n.s.*	*n.s.*	*n.s.*	*n.a.*

Note. n.a. not available. *n.s.* not significant. Pos – positive ES. Neg – negative ES. Medication moderator: 1 – medicated; 2 – not medicated; 3 – washout period of 24 h; 4 – washout period of 48 h. Comorbidity moderator: 1 – yes, comorbid disorder present; 2 – no, no comorbid disorder present. Cognitive function moderator: 1 – Inhibition; 2 – Attention; 3 – Working memory; 4 – Error processing. Modality moderator: 1 – visual; 2 – auditory; 3 – multimodal.

3.6. Publication bias analyses

Trim-and-fill analyses calculated to test for publication bias, revealed significant results for the following ERP components: Cue-P300-amplitude, Go-P200-latency, NoGo-P100-latency, NoGo-N100-latency, NoGo-P300-amplitude, CNV-amplitude, ERN-amplitude, ERN-latency, Pe-amplitude, and Pe-latency. For the NoGo-P300-amplitude, a smaller but still significant effect size emerged when an estimated number of 10 missing studies was imputed, indicating a potential publication bias (d = -0.30 [-0.48 – (-0.11)]). For the other ERP components, no significant results were obtained.

4. Discussion

4.1. Summary of effects: cognitive ERPs as brain-based biomarkers for ADHD

The current meta-analysis shows significant group-level ERP differences between ADHD and non-ADHD, most prominently in later components. The results indicate that individuals with ADHD show on average smaller Cue-P300-amplitudes, longer Go-P300-latencies, smaller NoGo-P300-amplitudes, longer NoGo-P300-latencies, smaller CNV-amplitudes, and smaller Pe-amplitudes compared to non-ADHD. In line with current theories on executive functioning deficits in ADHD (Kofler et al., 2019), the moderate to large effects obtained for these later components indicate core deficits in later, higher-order cognitive processing stages and might represent possible biomarkers of ADHD. Although, a potential publication bias might confound the results obtained for the NoGo-P300-amplitude analyses, the findings of both sensitivity analyses further support the idea that P300-components are the most sensitive ADHD-biomarkers.

Unexpectedly, individuals with ADHD also had shorter P100-latencies than non-ADHD. A possible explanation may be that in cognitive paradigms, the later part of the P100 includes higher involvement of cognitive modulation-processes. Therefore, shorter P100-latencies might be interpreted as a failure to further engage in such attentional processing necessary for successful cognitive modulation of sensory processing (Leroy et al., 2018).

Another unanticipated finding was that the current meta-analysis could not reliably confirm between-group differences for the N200-component. As outlined previously, heterogeneous results have been obtained for N200-alterations in ADHD in primary studies – a finding also reflected by the significant heterogeneity indices in the current meta-analysis. As can be seen from the moderator analyses, several demographic characteristics such as age, IQ, or comorbid disorders might influence the sensitivity of the N200 as a neurophysiological marker of ADHD. Therefore, the N200-component might indeed be relevant for the characterization of subgroups of individuals with ADHD (e.g. different age groups, IQ levels, with different comorbidities). In addition, non-significant overall results were obtained for the remaining ERP components: Cue-P300-latency, Go-P100-amplitude, Go-N100-amplitude and latency, Go-P200-amplitude and latency, Go-P300-amplitude, NoGo-P100-amplitude and latency, NoGo-N100-amplitude and latency, NoGo-P200-amplitude and latency, ERN-amplitude and latency, as well as the Pe-latency.

Furthermore, the current meta-analyses aimed at addressing sources of heterogeneity and, to this end, investigated several demographic and methodological characteristics. The moderator analysis for age group revealed stronger effects in children compared to adolescents or adults, for the P100, the N200, the P300, and the Pe components in different

Table 5c
Summary of meta-analytic findings for **latency** moderator analyses (mixed-effects models fitted) – CNV, ERN/Ne, Pe.

Latency	*k*	$Q_B(df, p)$	$Q_W(df, p)$	*Comparison*
CNV				
age	*n.a.*	*n.a.*	*n.a.*	*n.a.*
IQ	*n.a.*	*n.a.*	*n.a.*	*n.a.*
medication	*n.a.*	*n.a.*	*n.a.*	*n.a.*
comorbidity	*n.a.*	*n.a.*	*n.a.*	*n.a.*
task	*n.a.*	*n.a.*	*n.a.*	*n.a.*
cogn. function	*n.a.*	*n.a.*	*n.a.*	*n.a.*
modality	*n.a.*	*n.a.*	*n.a.*	*n.a.*
ISI	*n.a.*	*n.a.*	*n.a.*	*n.a.*
electrodes	*n.a.*	*n.a.*	*n.a.*	*n.a.*
ERN/Ne				
age	*n.s.*	*n.s.*	*n.s.*	*n.a.*
IQ	*n.s.*	*n.s.*	*n.s.*	*n.a.*
medication	*n.s.*	*n.s.*	*n.s.*	*n.a.*
comorbidity	*n.s.*	*n.s.*	*n.s.*	*n.a.*
task	*n.s.*	*n.s.*	*n.s.*	*n.a.*
cogn. function	*n.a.*	*n.a.*	*n.a.*	*n.a.*
modality	*n.a.*	*n.a.*	*n.a.*	*n.a.*
ISI	*n.s.*	*n.s.*	*n.s.*	*n.a.*
electrodes	*n.s.*	*n.s.*	*n.s.*	*n.a.*
Pe				
age	*n.s.*	*n.s.*	*n.s.*	*n.a.*
IQ	*n.s.*	*n.s.*	*n.s.*	*n.a.*
medication	8	7.28 (3, .06)	0.48 (5, 0.99)	4 < 3 (pos) < 1* (neg)
comorbidity	*n.s.*	*n.s.*	*n.s.*	*n.a.*
task	*n.s.*	*n.s.*	*n.s.*	*n.a.*
cogn. function	*n.a.*	*n.a.*	*n.a.*	*n.a.*
modality	*n.a.*	*n.a.*	*n.a.*	*n.a.*
ISI	8	6.83 (1, .009)	0.54 (6, 1.00)	*n.a.*
electrodes	*n.s.*	*n.s.*	*n.s.*	*n.a.*

Note. n.a. not available. *n.s.* not significant. Pos – positive ES. Neg – negative ES. Medication moderator: 1 – medicated; 2 – not medicated; 3 – washout period of 24 h; 4 – washout period of 48 h. Comorbidity moderator: 1 – yes, comorbid disorder present; 2 – no, no comorbid disorder present. Cognitive function moderator: 1 – Inhibition; 2 – Attention; 3 – Working memory; 4 – Error processing. Modality moderator: 1 – visual; 2 – auditory; 3 – multimodal.

task conditions. This finding is in line with previous literature (e.g. Johnstone et al., 2007), and might reflect a possible reduction of ADHD symptoms during adolescence and early adulthood, which is reported to occur in approximately 40 %–60 % of individuals with ADHD, primarily for symptoms of hyperactivity (Faraone et al., 2006). However, not all cognitive ERP alterations were reduced in adults. Consistent with results from Doehnert and colleagues (Doehnert et al., 2010), the CNV-amplitude that was reduced in ADHD showed no significant developmental effects, and could therefore be interpreted as a stable neurophysiological marker independent of age. For the Cue-P300-amplitude age-moderator analysis larger group differences were identified for adults compared to children or adolescents, indicating that the Cue-P300-component represents a neuromarker candidate for adult ADHD. Similar meta-analytic results were obtained from Szuromi and colleagues (Szuromi et al., 2011) who identified the Go-P300-component as a brain-based marker for ADHD in adults. As the obtained moderator effects might also result from a different number of studies included per age subgroup, they should be interpreted with caution. Nevertheless, age represents an important moderator that helps to understand phenotypic changes in the developmental course of ADHD. Age-related changes might primarily occur for later ERPs due to more efficient higher-order cognitive processing, reflected by a normalization of ERP amplitudes and latencies during the transition from childhood into early adulthood. Further studies need to explore how these results fit with models of prefrontal brain maturation in healthy, as well as ADHD populations. Regarding IQ, the respective moderator analysis indicated larger between-group differences for higher IQ values. Generally, primary studies emphasize the protective role of IQ in the developmental course of psychiatric disorders and for predicting a positive treatment response, suggesting *buffering* effects of higher intellectual abilities (Handen et al., 1997; Owens et al., 2003). Within the current meta-analysis, larger between-group differences were identified for higher intellectual abilities (mean across groups). One might assume that these larger group differences might be due to a lower ability of individuals with ADHD with higher intellectual abilities to exploit those capacities. Further studies are warranted to explore IQ-effects in more detail. The explorative moderator analysis for ADHD medication-status revealed very heterogeneous results. For some components, larger effects have been obtained in non-medicated ADHD and ADHD after a washout of medication compared to medicated ADHD (e.g. Pe-amplitude) – in line with previous literature reporting on a neurophysiological normalization in individuals with ADHD on appropriate medication (Taylor et al., 1993). Regarding other ERP components (e.g. Cue- and NoGo-P300-amplitude), results are mixed and indicate even more elevated between-group differences between medicated individuals with ADHD and non-ADHD as compared to unmedicated ADHD or ADHD after a medication-washout period and non-ADHD. These ERPs might be unaffected by medication and the medicated ADHD might represent those more severely affected, resulting in larger neurophysiological differences. Furthermore, as shown in previous studies, there are substantial neurophysiological differences between medication responders and non-responders that might help explaining the current results (Sunohara et al., 1997). However, for most of the included studies the information on the type of medication, dosing, and medication response is lacking and could not be explored. For comorbidity, the results present *dilution*, as well as *elevation* effects: the presence of comorbid conditions might result in even smaller or even larger between-group differences – presumably depending on the different types of comorbidities in individuals with ADHD (Rothenberger et al., 2000). Nevertheless, as for most of the studies the explicit type of comorbidity is not reported, no detailed analyses could be conducted. For the task-moderator analysis, four tasks revealed large effect sizes, reflecting substantial neurophysiological alterations in individuals with ADHD compared to non-ADHD: the CPT, the CPT-Flanker version, the Go/NoGo, and the Oddball task. This might partly be due to the popularity of these tasks and, consequently, large amount of studies using these tasks. Furthermore, as some of these tasks might also involve vigilance/sustained attention (e.g. Oddball task), the larger effect sizes might be due to further deficits in sustained attention in ADHD (Barkley, 1997). When effect sizes were compared for the different cognitive functions (inhibitory control, (selective) attention, working memory, and error processing), the largest effects emerged for inhibitory control. Inhibitory control has been reported previously in numerous studies as being particularly deficient in ADHD (Albrecht et al., 2005; Barkley, 1997; Oosterlaan et al., 1998; Quay, 1997; Sergeant, 2000, 2005). Future studies are warranted to explore this moderator effect in relation to developmental effects along the lifespan. Further task-related moderators have been explored, such as stimulus modality: results show that largest effect sizes were obtained for auditory stimuli. This finding is somewhat surprising as many studies on ERPs in ADHD use tasks with stimuli being presented visually. A possible explanation might be that visually presented stimuli are more salient and therefore, capture more attention, partly compensating vigilance and state regulation deficits in ADHD. Stimuli presented via different modalities are processed in different brain regions, thereby activating different neural generators. Depending on the electrode positions used for calculating ERP amplitudes and latencies, some neural generators might have more impact on the neurophysiological signal assessed than others, thereby yielding substantial between-study differences. This points out to the importance of conducting further studies using auditory stimuli compared to visual stimuli to explore the relationship between stimulus modality and neurophysiological deficits in ADHD in more detail taking into account the electrode positions used for calculating ERPs. Regarding the ISI, meta-analytic results show that for a longer time window between the presentation of each stimulus, group differences become

more elevated. This result might be interpreted as reflecting difficulties in awaiting the next stimulus presentation in the ADHD group, thereby indicating higher levels of impulsivity symptoms. For the number of electrodes used to assess ERPs, heterogeneous results were obtained in the respective moderator analyses. Further studies are needed to explore if more electrodes might be associated with higher sensitivity in detecting neurophysiological group differences between ADHD and non-ADHD.

4.2. Practical implications: limited utility of cognitive ERPs for diagnostic purpose, selection of individualized treatment strategies, and tracking of therapy outcomes in ADHD

Although the current meta-analyses identified later ERPs as possible markers of ADHD, results were characterized by substantial heterogeneity, not meeting criteria for clinical application of ERP-markers for diagnostic purpose on an individual level. The heterogeneity in effect sizes, and a number of other factors limit the practical implication of the results. This heterogeneity on a basic neurophysiological level (e.g. Lenartowicz and Loo, 2014) reflects the "*inescapable heterogeneity*" of the ADHD phenotype (Arns and Gordon, 2014). The substantial amount of variation in the distribution of effect sizes suggests the influence of further relevant moderator variables, such as varying clinical profiles, diversity of psychiatric comorbidities, varying patterns of neurocognitive impairment, and varying confounds by developmental effects (e.g. Aasen et al., 2018). More studies are needed to understand this heterogeneity, and to validate relevant ERP variables for multimodal classification approaches (Mueller et al., 2011). In addition, to further explore the sensitivity of ERPs as ADHD biomarkers, the question of how specific these neuromarkers are needs to be addressed (Thome et al., 2012): further studies are needed comparing different ADHD (sub-) groups, as well as individuals with ADHD with different types of comorbid symptoms to non-ADHD (Sur and Sinha, 2009). Additionally, machine-learning approaches might use ERPs for identifying ADHD subgroups based on the combination of diagnostic information from different modalities. Beyond that, future studies should try to link ERPs to continuous symptom dimensions adopting the RDoc approach.

Prior studies have noted the relationship between neurophysiological processes and therapy response to medication, as well as non-medication therapies (e.g. Banaschewski and Brandeis, 2007; atomoxetine: Yamamuro et al., 2016a; stimulants: Ogrim et al., 2016; neurofeedback and methylphenidate: Janssen et al., 2016a, b; slow-cortical potentials neurofeedback: Heinrich et al., 2004), indicating that ERPs might be useful as objective diagnostic add-ons that are easy to assess in a non-invasive way to predict and track therapy outcome. The current meta-analysis suggests to (further) explicitly test the predictive value of later ERPs as neuromarkers in a personalized medicine framework (we are aware of a few already published, as well as ongoing studies using EEG/ERPs to predict response to different therapeutic interventions; e.g. Ogrim et al., 2014; ESCAlife trial, Döpfner et al., 2017).

4.3. Limitations & future directions

A few limitations of the current meta-analysis need to be acknowledged. Generally, because of a small number of studies included for some of the ERP components[10], the results of the respective analyses should be interpreted with caution. Consequently, there is an urgent need for further studies exploring ERPs in ADHD. Although the current meta-analyses show substantial differences in later cognitive ERP components, further studies are warranted.

Within the current work we did not include any unpublished data. The inclusion of unpublished data could possibly itself introduce bias as the unpublished studies located might be an unrepresentative sample of unpublished work and as the studies might be of lower methodological quality (Higgins et al., 2019). Nevertheless, the current results might be slightly biased. To address this issue, publication bias analyses were calculated and reported. For reliably identifying biomarkers we are in clear need of further replication studies. Open science might be a desirable framework promoting such efforts. An open science approach might reduce publication bias, thereby facilitating future meta- and mega-analyses.

Due to the low number of studies included for some of the relevant ERPs (especially, for earlier ERPs), moderator variables had to be explored separately. For higher validity of results and exploring the interplay between influence variables, different moderators would have been included in one (full) model. Therefore, the results are explorative and need to be interpreted with caution. For some of the moderators, the number of studies included per subgroup varies substantially, rendering the comparison of categories less stable. As a consequence, future studies are needed to fill the gaps of knowledge on some of the moderator categories: first, there are only a few studies conducted on adolescents with ADHD. Second, most of the studies are conducted in males – a characteristic pattern obtained for studies on (psychiatric) disorders with a higher prevalence in males compared to females (Polanczyk et al., 2007). Most of the studies are conducted on individuals with ADHD of the combined subtype and further studies are warranted on ADHD inattentive and hyperactive/impulsive subtypes (Tamayo-Orrego et al., 2015). Therefore, no moderator analysis could be conducted on ADHD subtype. In addition, there is an urgent need for studies reporting on comorbid symptoms in individuals with ADHD, as well as medication status (possibly, plus adherence and medication response). Furthermore, one important research question remains unanswered at present: how does symptom severity influence effect sizes (Yamamuro et al., 2016b)? This question is highly relevant, especially as the changeability of ERP components according to the clinical phenotype is an important criterion for the validity of biomarkers. Due to a lack of reported information and a variety of different scales used to assess ADHD symptom severity, this highly relevant moderator variable could not be explored. Consequently, there is an urgent need for standardization of ADHD scales in research to compare results obtained from different studies.

Many more moderators might be potentially relevant for ERP-differences between individuals with and without ADHD (e.g. child- versus adult-onset ADHD, electrode location/signal generators). Due to the small number of studies for some of the ERPs, a lack of reporting in primary studies, and the many fine differences in the methodological implementation of the primary studies, we need further studies to explore the heterogeneity in effect sizes in more detail.

4.4. Conclusions

This is the first meta-analysis quantitatively summarizing relevant literature on cognitive event-related potentials (ERPs) in ADHD across the lifespan. In line with current executive functioning-deficit theories of ADHD, the findings confirm that, on a group level, ADHD is associated with specific neurophysiological alterations during cognitive tasks, particularly during later cognitive processing-stages. Compared to non-ADHD, individuals with ADHD show moderate differences, mainly regarding later cognitive ERP components (P300, CNV, Pe). Further studies are needed to fully understand the heterogeneity in effect sizes and the influence of moderator variables to clarify the potential of cognitive ERPs for supporting objective ADHD diagnosis and neurophysiological subtyping, for selecting individualized treatment strategies, and for tracking therapy outcomes. Clearly, identification of conditions ensuring larger effect sizes are needed before ERPs can

[10] $1 < k \leq 15$: Cue-P300-latency, Go-P100-amplitude & latency, Go-N100-amplitude & latency, Go-P200-latency, NoGo-P100-amplitude, NoGo-N100-amplitude, NoGo-P200-amplitude, NoGo-P100-latency, NoGo-N100-latency, NoGo-P200-latency, NoGo-N200-latency, NoGo-P300-latency, ERN/Ne-latency, and Pe-latency.

become helpful, objective tools supporting diagnostic stratification and precision medicine.

Declaration of Competing Interest

Tobias Banaschewski has served in an advisory or consultancy role for Actelion, Hexal Pharma, Lilly, Medice, Novartis, Oxford outcomes, Otsuka, PCM Scientific, Shire and Viforpharma. He received conference support or speaker's fee by Medice, Novartis and Shire. He is/has been involved in clinical trials conducted by Shire and Viforpharma. He received royalties from Hogrefe, Kohlhammer, CIP Medien, and Oxford University Press. Daniel Brandeis serves as an unpaid scientific consultant for an EU-funded neurofeedback trial. All other authors report no potential conflict of interest. The present work is unrelated to the above grants and relationships.

Acknowledgements

The current work was supported by the research consortium on ADHD, ESCA-Life, funded by the German Federal Ministry of Education and Research (FKZ 01EE1408E).

Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.neubiorev.2020.01.019.

References

Aasen, I.E., Ørgrim, G., Kropotov, J., Brunner, J.F., 2018. Methylphenidate selectively modulates one sub-component of the no-go P3 in pediatric ADHD medication responders. Biol. Psychol. 134, 30–38. https://doi.org/10.1016/j.biopsycho.2018.02.011.

Albrecht, B., Banaschewski, T., Brandeis, D., Heinrich, H., Rothenberger, A., 2005. Response inhibition deficits in externalizing child psychiatric disorders: an ERP-study with the Stop-task. Behav. Brain Funct. 1 (1), 22.

Arns, M., Gordon, E., 2014. Quantitative EEG (QEEG) in psychiatry: diagnostic or prognostic use? Clin. Neurophysiol. 125 (8), 1504–1506.

Banaschewski, T., Brandeis, D., 2007. Annotation: what electrical brain activity tells us about brain function that other techniques cannot tell us–a child psychiatric perspective. J. Child Psychol. Psychiatry 48 (5), 415–435.

Barkley, R.A., 1997. Behavioral inhibition, sustained attention, and executive functions: constructing a unifying theory of ADHD. Psychol. Bull. 121 (1), 65.

*Barry, R.J., Johnstone, S.J., Clarke, A.R., 2003. A review of electrophysiology in attention-deficit/hyperactivity disorder: II. Event-related potentials. Clin. Neurophysiol. 114 (2), 184–198.

Biederman, J., Faraone, S., 2005. Attention-deficit hyperactivity disorder. Lancet 366 (9481), 237–248.

Borenstein, M., Hedges, L.V., Higgins, J.P., Rothstein, H.R., 2010. A basic introduction to fixed-effect and random-effects models for meta-analysis. Res. Synth. Methods 1 (2), 97–111.

Bresnahan, S.M., Anderson, J.W., Barry, R.J., 1999. Age-related changes in quantitative EEG in attention-deficit/hyperactivity disorder. Biol. Psychiatry 46 (12), 1690–1697.

Cheng, C.H., Chan, P.-Y.S., Hsieh, Y.-W., Chen, K.-F., 2016. A meta-analysis of mismatch negativity in children with attention deficit-hyperactivity disorders. Neurosci. Lett. 612, 132–137.

Coles, M.G.H., Rugg, M.D., 1995. ERPs: an introduction. In: Rugg, M.D., Coles, M.G.H. (Eds.), Electrophysiology of Mind. University Press, Oxford, pp. 1–25.

Cortese, S., 2012. The neurobiology and genetics of attention-deficit/hyperactivity disorder (ADHD): what every clinician should know. Eur. J. Paediatr. Neurol. 16 (5), 422–433.

Doehnert, M., Brandeis, D., Imhof, K., Drechsler, R., Steinhausen, H.-C., 2010. Mapping attention-deficit/hyperactivity disorder from childhood to adolescence—no neurophysiologic evidence for a developmental lag of attention but some for inhibition. Biol. Psychiatry 67 (7), 608–616.

Döpfner, M., Hautmann, C., Dose, C., Banaschewski, T., Becker, K., Brandeis, D., et al., 2017. ESCAschool study: trial protocol of an adaptive treatment approach for school-age children with ADHD including two randomised trials. BMC Psychiatry 17 (1), 269.

Faraone, S.V., Biederman, J., Mick, E., 2006. The age-dependent decline of attention deficit hyperactivity disorder: a meta-analysis of follow-up studies. Psychol. Med. 36 (2), 159–165.

Faraone, S.V., Bonvicini, C., Scassellati, C., 2014. Biomarkers in the diagnosis of ADHD–promising directions. Curr. Psychiatry Rep. 16 (11), 497.

Gamma, A., Kara, O., 2016. Event-related potentials for diagnosing children and adults with ADHD. J. Atten. Disord 1087054716631821.

Handen, B.L., Janosky, J., McAuliffe, S., 1997. Long-term follow-up of children with mental retardation/borderline intellectual functioning and ADHD. J. Abnorm. Child Psychol. 25 (4), 287–295.

Hedges, L.V., Olkin, L., 1985. Statistical Methods for Meta-Analysis. Academic Press, Orlando, FL.

Heinrich, H., Gevensleben, H., Freisleder, F.J., Moll, G.H., Rothenberger, A., 2004. Training of slow cortical potentials in attention-deficit/hyperactivity disorder: evidence for positive behavioral and neurophysiological effects. Biol. Psychiatry 55 (7), 772–775.

Cochrane Handbook for Systematic Reviews of Interventions Version 6.0 (Updated July 2019). In: Higgins, J.P.T., Thomas, J., Chandler, J., Cumpston, M., Li, T., Page, M.J., Welch, V.A. (Eds.), Available from. www.training.cochrane.org/handbook.

*Janssen, T., Geladé, K., van Mourik, R., Maras, A., Oosterlaan, J., 2016a. An ERP source imaging study of the oddball task in children with attention deficit/hyperactivity disorder. Clin. Neurophysiol. 127 (2), 1351–1357.

Janssen, T.W.P., Bink, M., Geladé, K., van Mourik, R., Maras, A., Oosterlaan, J., 2016b. A randomized controlled trial investigating the effects of neurofeedback, methylphenidate, and physical activity on event-related potentials in children with attention-deficit/hyperactivity disorder. J. Child Adolesc. Psychopharmacol. 26 (4), 344–353.

Johnstone, S.J., Dimoska, A., Smith, J.L., Barry, R.J., Pleffer, C.B., Chiswick, D., Clarke, A.R., 2007. The development of stop-signal and Go/Nogo response inhibition in children aged 7–12 years: performance and event-related potential indices. Int. J. Psychophysiol. 63 (1), 25–38.

Johnstone, S.J., Barry, R.J., Clarke, A.R., 2013. Ten years on: a follow-up review of ERP research in attention-deficit/hyperactivity disorder. Clin. Neurophysiol. 124 (4), 644–657.

Kofler, M.J., Irwin, L.N., Soto, E.F., Groves, N.B., Harmon, S.L., Sarver, D.E., 2019. Executive functioning heterogeneity in pediatric ADHD. J. Abnorm. Child Psychol. 47 (2), 273–286.

Lenartowicz, A., Loo, S.K., 2014. Use of EEG to diagnose ADHD. Curr. Psychiatry Rep. 16 (11), 498.

Leroy, A., Petit, G., Zarka, D., Cebolla, A.M., Palmero-Soler, E., Strul, J., Dan, B., Verbanck, P., Chéron, G., 2018. EEG dynamics and neural generators in implicit navigational image processing in adults with ADHD. Neuroscience 373, 92–105.

Lesesne, C., Abramowitz, A., Perou, R., Brann, E., 2000. Attention Deficit/Hyperactivity Disorder: A Public Health Research Agenda. Centers for Disease Control and Prevention, Atlanta, GA.

Loo, S.K., Cho, A., Hale, T.S., McGough, J., McCracken, J., Smalley, S.L., 2013. Characterization of the theta to beta ratio in ADHD: identifying potential sources of heterogeneity. J. Atten. Disord. 17 (5), 384–392.

Luck, S.J., Heinze, H.J., Mangun, G.R., Hillyard, S.A., 1990. Visual event- related potentials index focused attention within bilateral stimulus arrays: II. Functional dissociation of P1 and N1 components. Electroencephalogr. Clin. Neurophysiol. 75 (6), 528–542.

Luck, S.J., Woodman, G.F., Vogel, E.K., 2000. Event-related potential studies of attention. Trends Cogn. Sci. (Regul. Ed.) 4 (11), 432–440.

Micoulaud-Franchi, J.A., Lopez, R., Vaillant, F., Richieri, R., El-Kaim, A., Bioulac, S., et al., 2015. Perceptual abnormalities related to sensory gating deficit are core symptoms in adults with ADHD. Psychiatry Res. 230 (2), 357–363.

Moher, D., Liberati, A., Tetzlaff, J., Altman, D.G., the PRISMA Group, 2009. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. Ann. Intern. Med. 151 (4), 264–269.

Mueller, A., Candrian, G., Grane, V.A., Kropotov, J.D., Ponomarev, V.A., Baschera, G.M., 2011. Discriminating between ADHD adults and controls using independent ERP components and a support vector machine: a validation study. Nonlinear Biomed. Phys. 5 (1), 5.

Nieuwenhuis, S., Ridderinkhof, K.R., Blom, J., Band, G.P., Kok, A., 2001. Error-related brain potentials are differentially related to awareness of response errors: evidence from an antisaccade task. Psychophysiology 38 (5), 752–760.

Ogrim, G., Kropotov, J., Brunner, J.F., Candrian, G., Sandvik, L., Hestad, K.A., 2014. Predicting the clinical outcome of stimulant medication in pediatric attention-deficit/hyperactivity disorder: data from quantitative electroencephalography, event-related potentials, and a go/no-go test. Neuropsychiatr. Dis. Treat. 10, 231.

Ogrim, G., Aasen, I.E., Brunner, J.F., 2016. Single-dose effects on the P3no-go ERP component predict clinical response to stimulants in pediatric ADHD. Clin. Neurophysiol. 127 (10), 3277–3287.

Oosterlaan, J., Logan, G.D., Sergeant, J.A., 1998. Response inhibition in AD/HD, CD, comorbid AD/HD+ CD, anxious, and control children: a meta-analysis of studies with the stop task. J. Child Psychol. Psychiatry 39 (3), 411–425.

Owens, E.B., Hinshaw, S.P., Kraemer, H.C., Arnold, L.E., Abikoff, H.B., Cantwell, D.P., et al., 2003. Which treatment for whom for ADHD? Moderators of treatment response in the MTA. J. Consult. Clin. Psychol. 71 (3), 540.

Pliszka, S.R., Liotti, M., Woldorff, M.G., 2000. Inhibitory control in children with attention-deficit/hyperactivity disorder: event-related potentials identify the processing component and timing of an impaired right-frontal response-inhibition mechanism. Biol. Psychiatry 48 (3), 238–246.

Polanczyk, G., De Lima, M.S., Horta, B.L., Biederman, J., Rohde, L.A., 2007. The worldwide prevalence of ADHD: a systematic review and metaregression analysis. Am. J. Psychiatry 164 (6), 942–948.

Polanczyk, G.V., Salum, G.A., Sugaya, L.S., Caye, A., Rohde, L.A., 2015. Annual Research Review: a meta-analysis of the worldwide prevalence of mental disorders in children and adolescents. J. Child Psychol. Psychiatry 56 (3), 345–365.

Polich, J., 2007. Updating P300: an integrative theory of P3a and P3b. Clin. Neurophysiol. 118 (10), 2128–2148.

Quay, H.C., 1997. Inhibition and attention deficit hyperactivity disorder. J. Abnorm.

Child Psychol. 25 (1), 7–13.
R Development Core Team, 2018. R: A Language and Environment for Statistical Computing. 2015: ISBN 3-900051-07-0: URL. R Foundation for Statistical Computing, Austria. http://www.R-project.org.
Rothenberger, A., Banaschewski, T., Heinrich, H., Moll, G.H., Schmidt, M.H., van't Klooster, B., 2000. Comorbidity in ADHD-children: effects of coexisting conduct disorder or tic disorder on event-related brain potentials in an auditory selective-attention task. Eur. Arch. Psychiatry Clin. Neurosci. 250 (2), 101–110.
Sergeant, J., 2000. The cognitive-energetic model: an empirical approach to attention-deficit hyperactivity disorder. Neurosci. Biobehav. Rev. 24 (1), 7–12.
Sergeant, J.A., 2005. Modeling attention-deficit/hyperactivity disorder: a critical appraisal of the cognitive-energetic model. Biol. Psychiatry 57 (11), 1248–1255.
Sunohara, G., Voros, J., Malone, M., Taylor, M., 1997. Effects of methylphenidate in children with attention deficit hyperactivity disorder: a comparison of event-related potentials between medication responders and non-responders. Int. J. Psychophysiol. 27 (1), 9–14.
Sur, S., Sinha, V., 2009. Event-related potential: an overview. Ind. Psychiatry J. 18 (1), 70.
Swanson, J., Sergeant, J., Taylor, E., Sonuga-Barke, E., Jensen, P., Cantwell, D., 1998. Attention-deficit hyperactivity disorder and hyperkinetic disorder. Lancet 351 (9100), 429–433.
Szuromi, B., Czobor, P., Komlósi, S., Bitter, I., 2011. P300 deficits in adults with attention deficit hyperactivity disorder: a meta-analysis. Psychol. Med. 41 (7), 1529–1538.
Tamayo-Orrego, L., Osorio Forero, A., Quintero Giraldo, L.P., Parra Sánchez, J.H., Varela, V., Restrepo, F., 2015. Differential effects of Attention deficit/hyperactivity disorder subtypes in event-related potentials. Rev. Colomb. Psiquiatr. 44 (2), 77–86.
Taylor, M., Voros, J., Logan, W., Malone, M., 1993. Changes in event-related potentials with stimulant medication in children with attention deficit hyperactivity disorder. Biol. Psychol. 36 (3), 139–156.
Taylor, M.J., Sunohara, G.A., Khan, S.C., Malone, M.A., 1997. Parallel and serial attentional processes in ADHD: ERP evidence. Dev. Neuropsychol. 13 (4), 531–539.
*Taylor, E., Döpfner, M., Sergeant, J., Asherson, P., Banaschewski, T., Buitelaar, J., et al., 2004. European clinical guidelines for hyperkinetic disorder–first upgrade. Eur. Child Adolesc. Psychiatry 13 (1), i7–i30.
Thome, J., Ehlis, A.-C., Fallgatter, A.J., Krauel, K., Lange, K.W., Riederer, P., et al., 2012. Biomarkers for attention-deficit/hyperactivity disorder (ADHD). A consensus report of the WFSBP task force on biological markers and the World Federation of ADHD. World J. Biol. Psychiatry 13 (5), 379–400.
Viechtbauer, W., 2010. Conducting meta-analyses in R with the metafor package. J. Stat. Softw. 36 (3).
Walter, W.G., Cooper, R., Aldridge, V., McCallum, W., Winter, A., 1964. Contingent negative variation: an electrical sign of sensorimotor association and expectancy in the human brain. Nature 203, 380–384.
*Yamamuro, K., Ota, T., Iida, J., Nakanishi, Y., Matsuura, H., Uratani, M., et al., 2016a. Event-related potentials reflect the efficacy of pharmaceutical treatments in children and adolescents with attention deficit/hyperactivity disorder. Psychiatry Res. 242, 288–294.
*Yamamuro, K., Ota, T., Iida, J., Nakanishi, Y., Suehiro, Y., Matsuura, H., et al., 2016b. Event-related potentials correlate with the severity of child and adolescent patients with attention deficit/hyperactivity disorder. Neuropsychobiology 73 (3), 131–138.
Yong-Liang, G., Robaey, P., Karayanidis, F., Bourassa, M., Pelletier, G., Geoffroy, G., 2000. ERPs and behavioral inhibition in a Go/No-go task in children with attention-deficit hyperactivity disorder. Brain Cogn. 43 (1-3), 215–220.

Further reading

*Albrecht, B., Brandeis, D., Uebel, H., Heinrich, H., Mueller, U.C., Hasselhorn, M., et al., 2008. Action monitoring in boys with attention-deficit/hyperactivity disorder, their nonaffected siblings, and normal control subjects: evidence for an endophenotype. Biol. Psychiatry 64 (7), 615–625.
*Albrecht, B., Brandeis, D., Uebel, H., Valko, L., Heinrich, H., Drechsler, R., et al., 2013. Familiality of neural preparation and response control in childhood attention deficit-hyperactivity disorder. Psychol. Med. 43 (9), 1997–2011.
*Baijot, S., Deconinck, N., Slama, H., Massat, I., Colin, C., 2013. Behavioral and neurophysiological study of attentional and inhibitory processes in ADHD-combined and control children. Acta Neurol. Belg. 113 (4), 477–485.
Barry, R.J., Clarke, A.R., McCarthy, R., Selikowitz, M., Brown, C.R., Heaven, P.C., 2009. Event-related potentials in adults with attention-deficit/hyperactivity disorder: an investigation using an inter-modal auditory/visual oddball task. Int. J. Psychophysiol. 71 (2), 124–131.
*Bluschke, A., Roessner, V., Beste, C., 2016. Specific cognitive–neurophysiological processes predict impulsivity in the childhood attention-deficit/hyperactivity disorder combined subtype. Psychol. Med. 46 (6), 1277–1287.
Cao, J., Wang, S., Ren, Y., Zhang, Y., Cai, J., Tu, W., et al., 2013. Interference control in 6–11 year-old children with and without ADHD: behavioral and ERP study. Int. J. Dev. Neurosci. 31 (5), 342–349.
*Chang, W.-P., Davies, P.L., Gavin, W.J., 2009. Error monitoring in college students with attention-deficit/hyperactivity disorder. J. Psychophysiol. 23 (3), 113–125.
*Cheung, C.H., McLoughlin, G., Brandeis, D., Banaschewski, T., Asherson, P., Kuntsi, J., 2017. Neurophysiological correlates of attentional fluctuation in attention-deficit/hyperactivity disorder. Brain Topogr. 30 (3), 320–332.
*Czobor, P., Kakuszi, B., Németh, K., Balogh, L., Papp, S., Tombor, L., Bitter, I., 2017. Electrophysiological indices of aberrant error-processing in adults with ADHD: a new region of interest. Brain Imaging Behav. 11 (6), 1616–1628.
*Dhar, M., Been, P.H., Minderaa, R.B., Althaus, M., 2010. Information processing differences and similarities in adults with dyslexia and adults with Attention Deficit Hyperactivity Disorder during a Continuous Performance Test: a study of cortical potentials. Neuropsychologia 48 (10), 3045–3056.
*Du Rietz, E., Cheung, C.H., McLoughlin, G., Brandeis, D., Banaschewski, T., Asherson, P., Kuntsi, J., 2016. Self-report of ADHD shows limited agreement with objective markers of persistence and remittance. J. Psychiatr. Res. 82, 91–99.
*Fallgatter, A.J., Ehlis, A.-C., Seifert, J., Strik, W.K., Scheuerpflug, P., Zillessen, K.E., et al., 2004. Altered response control and anterior cingulate function in attention-deficit/hyperactivity disorder boys. Clin. Neurophysiol. 115 (4), 973–981.
*Fisher, T., Aharon-Peretz, J., Pratt, H., 2011. Dis-regulation of response inhibition in adult Attention Deficit Hyperactivity Disorder (ADHD): an ERP study. Clin. Neurophysiol. 122 (12), 2390–2399.
*Grane, V.A., Brunner, J.F., Endestad, T., Aasen, I.E.S., Kropotov, J., Knight, R.T., Solbakk, A.-K., 2016. ERP correlates of proactive and reactive cognitive control in treatment-naive adult ADHD. PLoS One 11 (7), e0159833.
*Groom, M.J., Bates, A.T., Jackson, G.M., Calton, T.G., Liddle, P.F., Hollis, C., 2008. Event-related potentials in adolescents with schizophrenia and their siblings: a comparison with attention-deficit/hyperactivity disorder. Biol. Psychiatry 63 (8), 784–792.
*Groom, M.J., Cahill, J.D., Bates, A.T., Jackson, G.M., Calton, T.G., Liddle, P.F., Hollis, C., 2010. Electrophysiological indices of abnormal error-processing in adolescents with attention deficit hyperactivity disorder (ADHD). J. Child Psychol. Psychiatry 51 (1), 66–76.
*Herrmann, M.J., Mader, K., Schreppel, T., Jacob, C., Heine, M., Boreatti-Hümmer, A., et al., 2010. Neural correlates of performance monitoring in adult patients with attention deficit hyperactivity disorder (ADHD). World J. Biol. Psychiatry 11, 457–464 2-2.
*Jonkman, L., Kemner, C., Verbaten, M., Van Engeland, H., Kenemans, J., Camfferman, G., et al., 1999. Perceptual and response interference in children with attention-deficit hyperactivity disorder, and the effects of methylphenidate. Psychophysiology 36 (4), 419–429.
*Jonkman, L., Kemner, C., Verbaten, M., Van Engeland, H., Camfferman, G., Buitelaar, J., Koelega, H., 2000. Attentional capacity, a probe ERP study: differences between children with attention-deficit hyperactivity disorder and normal control children and effects of methylphenidate. Psychophysiology 37 (3), 334–346.
*Jonkman, L., Kenemans, J., Kemner, C., Verbaten, M., Van Engeland, H., 2004. Dipole source localization of event-related brain activity indicative of an early visual selective attention deficit in ADHD children. Clin. Neurophysiol. 115 (7), 1537–1549.
*Jonkman, L.M., van Melis, J.J., Kemner, C., Markus, C.R., 2007. Methylphenidate improves deficient error evaluation in children with ADHD: an event-related brain potential study. Biol. Psychol. 76 (3), 217–229.
*Karayanidis, F., Robaey, P., Bourassa, M., de Koning, D., Geoffroy, G., Pelletier, G., 2000. ERP differences in visual attention processing between attention-deficit hyperactivity disorder and control boys in the absence of performance differences. Psychophysiology 37 (3), 319–333.
*Kim, S., Liu, Z., Glizer, D., Tannock, R., Woltering, S., 2014. Adult ADHD and working memory: neural evidence of impaired encoding. Clin. Neurophysiol. 125 (8), 1596–1603.
*Linden, M., Gevirtz, R., Isenhart, R., Fisher, T., 1996. Event related potential of subgroups of children with attention deficit hyperactivity disorder and the implications for EEG biofeedback. J. Neurother. 1 (3), 40–49. https://doi.org/10.1300/J184v01n03_06.
*López, V., Lopez-Calderon, J., Ortega, R., Kreither, J., Carrasco, X., Rothhammer, P., et al., 2006. Attention-deficit hyperactivity disorder involves differential cortical processing in a visual spatial attention paradigm. Clin. Neurophysiol. 117 (11), 2540–2548.
Marzinzik, F., Wahl, M., Krüger, D., Gentschow, L., Colla, M., Klostermann, F., 2012. Abnormal distracter processing in adults with attention-deficit-hyperactivity disorder. PLoS One 7 (3), e33691.
*Mayer, K., Wyckoff, S.N., Schulz, U., Strehl, U., 2012. Neurofeedback for adult attention-deficit/hyperactivity disorder: investigation of slow cortical potential neurofeedback—preliminary results. J. Neurother. 16 (1), 37–45.
*McLoughlin, G., Albrecht, B., Banaschewski, T., Rothenberger, A., Brandeis, D., Asherson, P., Kuntsi, J., 2009. Performance monitoring is altered in adult ADHD: a familial event-related potential investigation. Neuropsychologia 47 (14), 3134–3142.
*McLoughlin, G., Albrecht, B., Banaschewski, T., Rothenberger, A., Brandeis, D., Asherson, P., Kuntsi, J., 2010. Electrophysiological evidence for abnormal preparatory states and inhibitory processing in adult ADHD. Behav. Brain Funct. 6 (1), 66.
*Michelini, G., Kitsune, G.L., Cheung, C.H., Brandeis, D., Banaschewski, T., Asherson, P., et al., 2016. Attention-deficit/hyperactivity disorder remission is linked to better neurophysiological error detection and attention-vigilance processes. Biol. Psychiatry 80 (12), 923–932.
*Perchet, C., Revol, O., Fourneret, P., Mauguière, F., Garcia-Larrea, L., 2001. Attention shifts and anticipatory mechanisms in hyperactive children: an ERP study using the Posner paradigm. Biol. Psychiatry 50 (1), 44–57.
*Rodriguez, P.D., Baylis, G.C., 2007. Activation of brain attention systems in individuals with symptoms of ADHD. Behav. Neurol. 18 (2), 115–130.
*Rommel, A.-S., James, S.-N., McLoughlin, G., Brandeis, D., Banaschewski, T., Asherson, P., Kuntsi, J., 2017. Association of preterm birth with attention-deficit/hyperactivity disorder–like and wider-ranging neurophysiological impairments of attention and inhibition. J. Am. Acad. Child Adolesc. Psychiatry 56 (1), 40–50.
*Senderecka, M., Grabowska, A., Gerc, K., Szewczyk, J., Chmylak, R., 2012a. Event-related potentials in children with attention deficit hyperactivity disorder: an investigation using an auditory oddball task. Int. J. Psychophysiol. 85 (1), 106–115.
*Senderecka, M., Grabowska, A., Szewczyk, J., Gerc, K., Chmylak, R., 2012b. Response

inhibition of children with ADHD in the stop-signal task: an event-related potential study. Int. J. Psychophysiol. 85 (1), 93–105.
*Smith, J.L., Johnstone, S.J., Barry, R.J., 2004. Inhibitory processing during the Go/NoGo task: an ERP analysis of children with attention-deficit/hyperactivity disorder. Clin. Neurophysiol. 115 (6), 1320–1331.
*Sokhadze, E.M., Baruth, J.M., Sears, L., Sokhadze, G.E., El-Baz, A.S., Williams, E.L., et al., 2012. Event-related potential study of attention regulation during illusory figure categorization task in ADHD, autism spectrum disorder, and typical children. J. Neurother. 16 (1), 12–31.
*Stroux, D., Shushakova, A., Geburek-Höfer, A., Ohrmann, P., Rist, F., Pedersen, A., 2016. Deficient interference control during working memory updating in adults with ADHD: an event-related potential study. Clin. Neurophysiol. 127 (1), 452–463.
*Sunohara, G.A., Malone, M.A., Rovet, J., Humphries, T., Roberts, W., Taylor, M.J., 1999. Effect of methylphenidate on attention in children with attention deficit hyperactivity disorder (ADHD): ERP evidence. Neuropsychopharmacology 21 (2), 218.
*Tsai, M.-L., Hung, K.-L., Lu, H.-H., 2012. Auditory event-related potentials in children with attention deficit hyperactivity disorder. Pediatr. Neonatol. 53 (2), 118–124.
*Tye, C., Asherson, P., Ashwood, K., Azadi, B., Bolton, P., McLoughlin, G., 2014. Attention and inhibition in children with ASD, ADHD and co-morbid ASD+ ADHD: an event-related potential study. Psychol. Med. 44 (5), 1101–1116.
*van der Stelt, O., van der Molen, M., Gunning, W.B., Kok, A., 2001. Neuroelectrical signs of selective attention to color in boys with attention-deficit hyperactivity disorder. Brain Res. Cogn. Brain Res. 12 (2), 245–264.
*Wild-Wall, N., Oades, R.D., Schmidt-Wessels, M., Christiansen, H., Falkenstein, M., 2009. Neural activity associated with executive functions in adolescents with attention-deficit/hyperactivity disorder (ADHD). Int. J. Psychophysiol. 74 (1), 19–27.
*Winsberg, B.G., Javitt, D.C., Shanahan, G., 1997. Electrophysiological indices of information processing in methylphenidate responders. Biol. Psychiatry 42 (6), 434–445.
*Woltering, S., Liu, Z., Rokeach, A., Tannock, R., 2013. Neurophysiological differences in inhibitory control between adults with ADHD and their peers. Neuropsychologia 51 (10), 1888–1895.
*Yorbik, O., Ozdag, M.F., Olgun, A., Senol, M.G., Bek, S., Akman, S., 2008. Potential effects of zinc on information processing in boys with attention deficit hyperactivity disorder. Prog. Neuropsychopharmacol. Biol. Psychiatry 32 (3), 662–667.
*Yorbik, Ö, Mutlu, C., Özdag, M.F., Olgun, A., Yilmaz, G., Ayta, S., 2016. Possible effects of copper and ceruloplasmin levels on auditory event potentials in boys with attention deficit hyperactivity disorder. Noro Psikiyatr. Ars. 53 (4), 321.